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AUGUST 1940

Volume I. Number 8.

DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

PUBLISHED MONTHLY for the PHYSICIAN in the GENERAL PRACTICE of MEDICINE

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VOL. 1 AUGUST 1940 NO. 8

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800 AVENUE B., GALVESTON, TEXAS

BUSINESS OFFICES
PHYSICIANS POSTGRADUATE PRESS
500 NORTH DEARBORN ST., CHICAGO, ILLINOIS

PRINTING OFFICES
ALAMOGORDO ROAD, EL PASO, TEXAS

DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

EDITORIAL COMMENT

Psychiatry's Contributions to the Philosophies of General Medicine

Up to the end of the nineteenth century a veil of mystery obscured the causative factors involved in mental disorders. Consequently, there was a welter of confusion, even in the concepts and classifications of these disorders. In its efforts to bring order out of chaos, psychiatry conceived the idea of taking the life history, so to speak, in an effort to find common denominators which might be the starting points for the development of maladjustments. In doing this it was found that no one factor could be held responsible, as was thought to be the case in diseases with which medicine proper dealt. There were a multiplicity of factors involved: those inherent in the individual and those in his moving, changing environment which acted upon him.

Out of this grew the concepts of the genetic-dynamic attitude: man as a fluid entity with his behavior, as seen at any one time, having its origin at certain points in certain circumstances and evolving into the present general pattern. Nothing taking place in the individual's life could be neglected, all having relative values. This included what is ordinarily thought of as "physical" circumstances as well as "psychological," the two being inseparable as a matter of fact. Out of this grew the concept of the "person as a whole".

Again it was found that the happenings in the person's environment, both physical and particularly personal, played a very strong role in determining behavior. This is true not only of his immediate environment, but also of the general "cultural pattern" in which he lives. Consequently, attention has to be paid not only to what is going on within the individual, but to what is going on outside.

All of this rightly has influenced general medical thought greatly. Up to this time medicine tended to get away from consideration of the person. It was becoming almost completely concerned with isolated structure and function. Psychiatry has influenced medicine to get back to an interest in the person who happens to house perverted structure and function. In addition, psychiatry has shown that the doctor cannot stop there, but must take into consideration the circumstances under which the person lives and moves.

This philosophy is beautifully brought out in Dr. Canby Robinson's book "The Patient as a Person" published by the Commonwealth Fund of New York. In this he brings out the necessity for considering the "person as a whole" and in addition the social factors involved in his living conditions. For instance: "112 patients were interviewed.....Of those 77 or 68.8 per cent, were found to present adverse social conditions needing study and treatment. In 52.6 per cent of the patients adverse social conditions affected their illness, and in 57.2 per cent they were related to the required treatment or to desirable adjustments of their mode of living." We cannot urge too strongly that all physicians read this book.

Consequently, medical therapy has taken on new breadth of vision. One cannot be concerned solely with isolated structural or functional difficulties but must be concerned with the individual. And this concern must take in the social structure of his environment so that the help of social agencies may be helpfully elicited.

W. W. Young, M.D.,

Atlanta, Georgia

A Review of Dermoid, Teratoid and Teratomatous Intracranial Tumors

WILLIAM H. SWEET, M.D.
Chicago, Illinois

Dr. Sweet took the research degree, B. Sc., of Oxford University under Professor Sherrington in 1934; received his M.D. from Harvard University in 1936. His postgraduate training has been taken largely at the University of Chicago Clinics with short periods at the Massachusetts General Hospital in Boston and the National Hospital for Nervous Diseases in London. He has been an instructor in neurosurgery at the University of Chicago Clinics for the past year.

Since these tumors occur so infrequently that even the largest clinics have seen only a few, there has never been a review of the literature of the group as a whole to see whether useful clinical information might emerge in a compact form. Such an attempt is made here. Two brief case reports are included.

We shall begin by differentiating the terms of the title. Upon reviewing the previous studies of these tumors we find that there is no generally accepted boundary dividing the so-called complex dermoid from the teratoid new growths. Kaufmann's Pathology (p. 1503, 1929) states that mixed tumors in which a cystic sac is the principal structure are complex dermoids. According to this conception any non-dermal elements in the tumors, as for example the frequently occurring bone or cartilage, are derived by metaplasia of the connective tissue elements of the skin. But Ewing (p. 1014-1018, 1928) states that the idea of a metaplasia has been replaced by the conception of a natural unfolding of embryonal cell potencies; that we now call many tumors teratomas which were formerly known as mixed tumors; and that, since the bulk of the early embryo is ectoderm, the overgrowth of this layer is a natural result. We know of no decisive evidence in favor of either of these viewpoints, but mention the conflict because of the confusion it has created. Hosoi (1930) and recently Weber (1939) have collected from the literature the teratoid and teratomatous intracranial tumors and Brock and Klenke have collected other cases reported as intracranial dermoids. Numerous tumors in each of these groups were mainly cystic and composed predominantly of dermal elements with small amounts of mesodermal tissue not seen in the normal corium of the skin. In fact, in Brock and Klenke's (1931) own case the tumor contained bone and they state that in terms of strictest definition such a growth belongs in the teratoid group.

We have collected 156 cases of dermoid, teratoid and teratomatous intracranial tumors reported in the literature under a variety of diagnoses. In considering the whole group it becomes apparent that these tumors consist pathologically of a series of gradually increasing complexity and that any separation of them into sub-groups must be at arbitrary points. We have classified them, in accordance with the majority opinion, using these criteria: (1) dermoids are tumors containing only elements found in the epidermis and corium; (2) teratoids are mixed tumors that contain elements of one or two embryonic germ layers including other tissues than those found in the skin; (3) teratomas contain elements of all three germ layers. Epidermoids, tumors containing only epidermis (but no hairs or glands), have not been included in this study, although numerous reports read during its progress indicate that their characteristics are essentially the same as those of the dermoids. The only major difference noted was the presence of a large group of epidermoids located in the cerebellopontine angle. Only one of the tumors in this study was so situated. For a recent review of the epidermoid tumors Mahoney (1936) may be consulted.

The hypophysial duct tumors (craniopharyngiomas), although they too arise from embryonal remnants, have not been included in the review. However, many of these tumors contain bone (see Bailey, Buchanan and Bucy, p. 365, 1939), and it is possible that such tumors belong to a transitional type between typical craniopharyngiomas and the tumors under discussion here.

The following features of clinical value appear from the review: *Age of appearance of symptoms.* Although the most widely accepted hypothesis regarding the etiology of these tumors is that they arise from pluri-potential cells which became misplaced or which failed

to develop in the embryo, symptoms do not appear in some cases until middle age. Twelve of the 156 cases, about 8 per cent, were over fifty years of age at the time the diagnosis was made.

Table II

Number of cases in each age group at time of diagnosis

Foetus	
10 yrs.	48
11-20 yrs.	39
21-30 yrs.	23
31-40 yrs.	17
41-50 yrs.	12
51-60 yrs.	7
61-70 yrs.	1
71-80 yrs.	4

It is interesting that Bailey, Buchanan and Bucy (p. 372, 1939) in a review of 138 cases of craniopharyngiomas, noted similar figures for the age distribution. For example, 10 per cent of those cases caused symptoms at over fifty years of age. The solid teratoids and teratomas occur almost exclusively in the first three decades. The tumors may grow so slowly that they are only incidental findings at autopsy. In this category are: (1) the walnut-sized teratoid in one lateral ventricle reported by Strassmann and Strecker (1887), (2) and (3) two walnut-sized dermoids, one at the apex of the left temporal lobe and the other (in another case) attached to the choroid plexus of the left lateral ventricle reported by Erdheim (1904), and (4) a 4x5 cm. dermoid in the left Sylvian fissure reported by Kopriwa (1927). The even more slowly growing epidermoids have given no symptoms during life in a greater number of cases.

Sex—There is a marked predominance of males in the series—94 as compared with 49 females, or a ratio of nearly 2 to 1.

Type of tumor—There are 44 teratomas, 50 teratoids and 62 dermoids.

Location—From Table III it is apparent that these tumors may be found almost anywhere in the cranial cavity, but that there are three sites of predilection, the pineal region, the pituitary region and the posterior part of the posterior cranial fossa. There are a great number of widely differing cellular types in the developing pineal and pituitary regions. In the former area there are paraphysial and epiphysial structures, mesoderm, dermal ectoderm and neuroectoderm, while in the latter locality there are mesoderm, notochord, neurectoderm and ectoderm from the stomodeum. Consequently, it is not surprising that the embryonal remanants in these regions form tumors which are almost always of complicated structure, either teratoid or teratomatous, with much solid tissue in them, and may be malignant as a result of rapid growth as well as relatively inaccessible and vital position.

The pineal tumors occur more frequently and are likely to be more complex histologically than those in the pituitary region. In addition to the cases recorded in Table I, there are extraordinary tumors of questionable cellular type occurring in the pineal area. Such neoplasms reported by Askanazy, Ziegler, Turner and Goldzieher have all been rapidly growing, and positive identification of the types of cells seems to have been difficult. (See the table of Bochner and Scarff, 1938). The interesting syndrome, macrogenitosomia praecox, found in some male children with neoplasms in the pineal area, but not in others, remains a mystery. Four major variations in the local pathogic status are possible. There may be: (1) a tumor in the pineal region not involving the pineal gland proper—merely pressing on it; (2) a pinealoma; (3) neoplastic destruction of the pineal gland by a tumor containing no pineal tissue; (4) neoplastic destruction of the pineal gland by a tumor containing adult or embryonic pineal tissue. In every one of the four groups there are both boys who have shown sexual precocity, and boys who have not shown it. (See Table I). Hence it seems impossible to correlate that syndrome with the status of the pineal tissue. Kahn is the only neurosurgeon to report the successful operative removal of a pineal teratoma.

The pituitary tumors of the types reported are almost as difficult from the surgical therapeutic standpoint. A small group of four tumors in the third ventricle have been so classified because it was impossible to determine an origin from the pineal or pituitary region. Probably they arise, as do the tumors in the other ventricles, from cells near or in the choroid plexus.

In the series, there are four colossal teratomas which replaced most of the brain. These

tumors, inconsistent with post-natal life, are probably much more numerous than this figure would indicate, since many of the cases have probably either been reported as monstrosities, or not reported at all.

The remainder of the groups present a much more hopeful outlook for the neurosurgeon and many cases of successful removals are now in the literature. Not only are they all in more accessible locations, but in all other areas the dermoids and cystic teratoids predominate strikingly over the surgically difficult, solid teratoids and teratomas. The simpler structure of these groups is doubtless due to the fact that dermal or neural ectoderm, or mesoderm are usually the only embryonic formations available. The most frequent site for these more easily removed pathologic types is the posterior part of the posterior cranial fossa near the midline, fortunately one of the most favorable locations for the neurosurgeon. Evidence that mesodermal and dermal ectodermal rests become trapped postero-inferior to the rhombencephalon is given not only by the location of the tumors themselves, but also by a developmental defect in the occipital bone in some of these cases.

Tumors in the orbital cavity and cranial fossae—Seven cases have had this curious location. To the six already in the literature we add another.

Case Report

Miss E. S., a forty-seven year old bookkeeper, entered Dr. Bailey's service at the University of Chicago Clinics on October 4, 1933. Four years previously she had noted the gradual onset of a left exophthalmus, diminishing visual acuity in the left eye, and pain over that eye. Following an ethmoidal operation and radium treatment to the eye elsewhere, she thought that these symptoms were all improved, but two years later she decided they were gradually recurring. X-rays of the skull at that time showed a sharply outlined left superior orbital fissure four times the size of the corresponding normal right fissure. This increase was produced by a moderate diminution in size of the lesser wing of the sphenoid and by a more pronounced decrease in size of the greater wing. By the time of her hospital entry, two years later, the sight in the left eye was gone, the exophthalmus had increased and there were severe pains above and lateral to the left orbit.

Twenty-three years previously she had had violent pains in the left side of the head and face and a stiff neck for nearly a year. There was double vision during that time. These symptoms may well have been caused by the same tumor.

On complete physical and neurological examination at entry her abnormal findings were:

(1) primary optic atrophy and no light perception, left eye;
(2) exophthalmometer reading: right 17 mm., left 22 mm.;
(3) right pupil, direct but no consensual reaction to light; left pupil, consensual but no direct reaction;
(4) 20 degree deviation of left eye in primary position;
(5) on elevation of eyes deviation of the left eye still further laterally.

There was no pulsation of the left eyeball and no bruit. X-rays of the skull showed little change from the films two years previously.

Operation: This was done October 7, 1933, by Dr. Bailey. A low left temporal bone flap with exposure of the posterior part of the orbit disclosed a brownish black cystic tumor. Part of it lay extradurally in the anterior and middle cranial fossae; another part extending into the orbit pushed the normal orbital contents medially; and a third portion filled the left maxillary sinus. Thirty cc. of milky brownish fluid, obtained upon puncture of the cyst, contained numerous cholesterol crystals, much hemosiderin and some leucocytes. The wall of the cyst was completely removed from all of the sites of its extension. Microscopically the wall consisted of a row of epithelium from one to three cells in thickness. A few small nerve cells and some chronically inflamed connective tissue lay external to the epithelium. In the connective tissue were many narrow (cholesterol) clefts lined by giant cells. The patient recovered promptly from the procedure, regained light perception in the left eye, and six years later presented no evidence of recurrence of her tumor.

The other cases of this type have been reported by de Lapersonne (1893), Raueiser (1919), Verebély (1929), Stender (1937), and Weber (1939). Every one of them has been cured by an operative removal of the tumor. Raueiser's and Weber's cases were teratoid cysts; all of the others were dermoids. Stender has advanced convincing reasons for believing that congenital failure of ossification of a portion of the orbital wall is responsible for the extraordinary hour-glass form taken by these tumors. He does not think they extend through normal fissures or foramina and then enlarge the opening by pressure. The sharp outline of the enormous superior orbital fissure in the x-rays of our case would confirm that view.

Duration of symptoms—As one would ex-

pect, the clinical course was usually measured in years. The longest recorded duration was in one of the cases of Rows (1906), a man of seventy-three years who had had convulsions for over thirty-eight years. The case of Farthing and Love (1937), had had headaches from her earliest childhood—for at least twenty years.

For three different reasons, however, there may be a sudden onset of symptoms. In the first type, the slowly growing tumor gives no evidence of its presence until there seems to be a relatively sudden breakdown in compensation of the mechanisms regulating intracranial pressure. The case of Sweet (1940), in which there was a sudden onset of severe symptoms and a duration of only ten weeks to the history at the time of the first hospital admission, is a typical example of this group. The same phenomenon has been seen with sufficient frequency in meningiomas to preclude a positive diagnosis of rapidly growing intracranial neoplasm on the basis of a short clinical history.

But in the present group of tumors there are two other causes for a sudden onset. In one type a malignant change may take place in one of the tissue elements composing the neoplasm with a correspondingly rapid evolution of symptoms. Seven cases in the series showed this (Maier, 1861, Falkson, 1879, Neumann, 1900, Bregmann and Steinhaus, 1907, Strohmeyer, 1910, Frank, 1920, Klapproth, 1922). These cases all progressed steadily to death within four to ten weeks from the clinical onset. In three of the cases the malignant changes were described as carcinomatous, in three of the cases as sarcomatous, and in one of the cases as occurring in the pineal parenchyma. Four of the seven tumors were in the pineal region, two were in the cerebral hemisphere and one was in the pituitary region. In two other sarcomatous pineal tumors (Coats, 1887, Oestreich and Slawyk, 1899) symptoms had been present for five months and three years, respectively. Perhaps the tumor had begun to produce symptoms before the sarcomatous change occurred.

There may be distinguished from the foregoing group another series of cases in which the onset of symptoms is more abrupt, suggesting a meningitis, or is even so sudden as to suggest a cerebral vascular accident. In these cases (Beneke, 1895, Winteler, 1910, Erdheim, 1904, Helly, 1925, Scheinker, 1936) the sudden onset of symptoms has been due to a rupture of the neoplastic cyst. One might expect that an outpouring of the cystic fluid into the cerebrospinal fluid would permit absorption of the former and that a salutary decrease in the size of the neoplasm would be the only result. Actually the cystic fluid, usually containing cholesterin, is highly irritative and is not absorbed. In the cases of Beneke and Winteler the rupture of the cysts brought about paralyses and aphasia so suddenly that cerebral hemorrhages were thought to have occurred; the patients died in a few days. In a case of Erdheim the patient, following trauma, ruptured a teratoid cyst into the third ventricle and died in a few hours. Erdheim does not indicate the severity of the primary accident, and hence we cannot be certain of the relative lethal roles of the accident and of the cystic contents. In the other cases the symptoms were not so instantaneous and overwhelming and not only the clinical course, but also the presence of many polymorphonuclear leucocytes in the spinal fluid suggested meningitis. In the case of Scheinker the patient's illness began with a severe headache; within three days he was unable to speak understandably or to execute commands; his spinal fluid cell count revealed 138 white cells; and in fourteen days he was dead. In Helly's case there had been an attack of "meningitis" at the age of sixteen years with complete recovery. Twenty years later similar symptoms reappeared — convulsive seizures with severe dyspnoea and nuchal rigidity—and within two weeks the patient died. In each of these cases the subarachnoid space was filled with lipoid material from a ruptured dermoid cyst. Bender (1925) has demonstrated experimentally by injections of various lipoid fractions from myelin into the subarachnoid space of rabbits that cholesterin is more irritative than the cerebrosides or glucosides, and gives rise to a leucocytic pleuocytosis in the spinal fluid that lasts for weeks. Greenfield (p. 1224, 1932) and Krieg (1936) have seen the same reaction in sterile spinal fluid following operative removals of these tumors in man. This reaction may become so pronounced that the patient's life is endangered. Rand (1925) has had such an experience; a sterile abscess developed at the site of a dermoid cyst which he had

only partially removed at the first operation, and the patient died. He thinks the entire cyst should probably have been removed at that first procedure. The cases in this group all serve to demonstrate that the contents of these cysts should be evacuated by the neurosurgeon without allowing any escape into the subarachnoid space, and that a complete removal of the cyst wall precluding further formation of these irritative substances is desirable.

Tendency to abscess formation within the cyst—In addition to sterile abscess formation there is a tendency for bacteria to find a nidus in the cysts and convert the cavity into an infected abscess. In Gladstone's (1937) case the bacteria probably made their way into the cyst by way of a tract extending to the surface through a midline defect in the occipital bone. In Todesco's (1927) infected dermoid of the right cerebellar hemisphere, a Gram positive diplococcus was cultured from the blood and spinal fluid. The infection probably reached the tumor by way of the blood stream from an infected hairy cyst over the external occipital protuberance. In Horrax's (1922) case 2, an abscess in an extradural intracranial dermoid cyst had been evacuated elsewhere and Cushing found another dermoid cyst in the right temporal lobe filled with greenish pus from which streptococci were recovered.

Case Report

A case of epidermoid cyst (Mr. F. D., aged forty-five years) seen in consultation by Dr. Bailey in April 1936, was found at post-mortem examination a few days later to have an abscess 6 cm. in diameter within the substance of the left frontal lobe. Streptococcus viridans and an enterococcus were cultured from the pus. On microscopic examination the abscess cavity was found to be lined by stratified squamous epithelium and it was then realized that an epidermoid cyst had become secondarily infected. No primary source of the infection was found elsewhere in the body, and there was no history of previous infection obtainable from the patient's wife.

Leaks from cysts in patients with a long history—The patients with dermoid cysts reported by Raymond et al (1904), Bathhurst (1895), Lua (1929) and Bauditz (1933), each showed contents from the main cyst scattered throughout much of the subarachnoid space and ventricular system. The lipoid globules became surrounded by giant cells and fixed in position, so it was evident that they had not been set free shortly before death, as in the group of cases discussed above. Furthermore, all four of these patients had had symptoms related to the cysts for several years prior to death, and three of them had a severe internal hydrocephalus involving all four ventricles. Presumably, the cellular reaction to the lipoid nodules in the leptomeninges had resulted in interference with the passage of cerebrospinal fluid. Either the cystic material leaked out more slowly, or was less irritative than that in the group of patients who were killed by rupture of their cysts. In Lua's case the spinal fluid at lumbar puncture during life showed both intra- and extracellular lipoid globules. In the case of Raymond et al., fat in the spinal fluid obtained before death stained black with osmic acid and blue with hematin as did the cystic contents post mortem.

Multiple tumors — Otto (1830), Heschl (1860), Bonorden (1892), Trachtenberg (1898), and Lua (1929), have each reported a case in which multiple intracranial dermoids were present, and Horrax (1922) has reported two cases in each of which two intracranial dermoids were present. In all but the cases of Otto and Trachtenberg, the tumors lay in the same general region, so the neurosurgeon has this added reason for re-exploring his original operative area thoroughly, if the patient does not recover in the expected fashion. This is exactly what was done in one of the cases of Horrax, and the second tumor was found. In the case of Kischensky and Tiesenhausen (1912) there was in addition to the main cerebellar teratoid tumor another pea-sized teratoid in the midline of the tentorium cerebelli. Several cases of multiple intracranial epidermoid tumor have also been reported.

Other congenital defects — The fault in embryonic development indicated by the presence of the tumor was accompanied in many cases by other congenital malformations. A glance at the "Remarks" in Table I will show what a wide variation of faults may be present concomitant with that inside the cranium.

Congenital defects overlying the tumor—The fault consisted usually of a defect in the cranial bone external to the tumor. In

Heschl's (1860) case a parietal dermoid cyst communicated with the scalp by way of a canal in the bone, and in J. Ogle's (1855) case a dermoid over the occipital lobe had a similar prolongation. In six other cases the defect was related to a tumor in the posterior part of the posterior cranial fossa. Of these, the cases of Gladstone (1937), Kwan (1930), Lannelongue and Achard (1886), and Ruschhaupt (1906) all had tumor extending into a canal in the occipital bone. The bony defect in the occiput in Cushing's (1925) case was observed in the clinical x-rays. In Todesco's (1927) case there was a small hairy cyst over the external occipital protuberance.

Roentgenologic evidence — Not only the above-mentioned bony defects in the skull, but also sufficient calcium in the bone, teeth or other tissues of these tumors to cast a shadow may localize this form of tumor. In the cases of Brock and Klenke (1931), Horrax (case 1, 1922), Heymann (1933), Rand (1925), Scott (1932) and Sweet (1940) such shadows were noted. The amount of bone present in the tumors of many of the other cases was probably sufficient to permit observation on good x-ray films had they been made.

When the tumors lie wholly or partially within one of the lateral ventricles a bizarre appearance may result at pneumencephalography. The air may enter between the nodules of the tumor and around the margins of its intraventricular portion. The ventricle is usually enlarged and one sees a collection of peculiarly disposed filaments of air instead of a recognizable ventricular shadow in the involved hemisphere. This has been observed several times in dermoid and epidermoid tumors. A reproduction in Krieg's (1936) report of his case 2 illustrates this.

Summary

The chief features of clinical value arising from the study of the literature of dermoid, teratoid and teratomatous intracranial tumors are:

1. Onset of symptoms usually occurs in the young, but may take place at any age.

2. The pineal region, the pituitary region and the posterior part of the posterior cranial fossa are the favorite sites for these tumors, but they may occur anywhere in the intracranial cavity. In the pituitary and pineal regions the tumors are usually solid, of complex histologic structure, and rarely removable. Elsewhere they are usually cystic, histologically simple, and many patients have been cured by operative removals.

3. The duration of symptoms is usually long, but some benign, easily removable types have histories of only a few weeks. If one of the tissues in the tumor undergoes malignant change, the evolution is rapid and, if the neoplastic cyst ruptures, the irritant fluid therefrom may kill the patient in a few hours to a few weeks. The cholesterin in the fluid seems to be the chief deleterious substance.

4. Even if the patient is not killed within a few weeks by rupture of his cyst a chronic cellular reaction to the lipoid globules in the ventricles and/or subarachnoid space may impede the circulation of the cerebrospinal fluid and a severe internal hydrocephalus develop. Fat has been found in the spinal fluid at lumbar puncture following rupture of dermoid cysts.

5. Infected abscesses may occur in the cysts prior to operation, and post-operatively there is a marked tendency to development of a sterile meningitis and/or abscess at the site of removal—hence the desirability of keeping the irritant, cholesterin-containing cystic contents away from the brain at operation, and of removing completely the neoplastic wall.

6. In all but two of the cases with multiple tumors the neoplasms were grouped close together in one area of the brain. Consequently, it is advisable to re-explore the original operative site if the patient does not recover appropriately from the first procedure.

7. Another congenital defect elsewhere in the body may be present along with the embryonic fault constituting the intracranial tumor.

8. A small congenital defect, a canal in the bone overlying the tumor (at times demonstrable by x-ray), or a cyst in the overlying scalp, is especially likely to be present.

9. Other roentgenologic evidence of the tumors may be given by calcified shadows within them. When the mass extends into an enlarged lateral ventricle, the penumencephalograms may show a bizarre scattering of air within the involved hemisphere.

10. An especially favorable group for operation is that in which an hour-glass form of

tumor lies both in the orbit and in the anterior or middle cranial fossa. All of the reported cases of this type have been cured by removal of the neoplasm.

Table III

Location of the Tumors

	Pituitary region	Pineal region	Posterior part of posterior cranial fossa	Replacing almost entire brain	Lateral ventricle	Third ventricle
Teratomas	4	28	3	4	1	1
Teratoids	11	11	7	0	3	1
Dermoids	1	1	17	0	4	2
Total	16	40	27	4	8	4

	Fourth ventricle	Orbital cavity and cranial fossae	Midbrain	Cerebello-pontine angle	Anywhere on inferior surface except pituitary region	Elsewhere over or in cerebral hemispheres
Teratomas	1	0	1	0	0	1
Teratoids	0	2	0	1	7	7
Dermoids	5	5	0	0	15	19
Total	6	7	1	1	22	27

Table I

Summary of Case Reports

The material herein contained is grouped as follows: Author, Date of Publication, Bibiliographical Reference, Age of Patient; Sex of Patient; Diagnosis Size of Tumor; and Remarks.

Verattus; 1745; *De Bononiensi scientiarum et artium instituto atque academia commentarii,* 2: Pt. 1, 184; 50 yr.; F; Dermoid pea.

Morgagni; 1764; *Epistolae Anatomicae Duodeviginti. Patavii,* Ep. XX, Art. 58, P. 396; Foetus; F; Dermoid "small"; Palatal defect and encephalocoele associated.

Otto; 1830; *Lehrbuch der pathologischen Anatomie.* Bd. 1, pp. 381, 419, 433; Newborn; Dermoids.

Clairat; 1838; *Gazette des Hopitaux 12:* 166; 30 yr.; F; Dermoid 161 gms.

Paget; 1853; *Lectures on Surgical Pathology.* Vol. II, *Tumours,* p. 85—London: Longman, Brown, Green and Longmans; "Old"; M; Dermoid "seed".

Ogle J.; 1855; *Tr. Path. Soc. London, 6:* 12-16; 2½ yr.; M; Dermoid 3.8x2.5 cm.; Communicating with scalp via bony canal.

Rokitansky; 1855; *Lehrbuch der Pathologischen Anatomie. Dritte Aufl. Wien, W. Braumüller,* Bd. 1 p. 221; 50 yr.; F; Dermoid bean.

Heschl; 1860; *Vierteljahrschrift f. d. praktische Heilkunde 68:* 49; Foeuts; Dermoids (four) largest 2.7x6.6 cm.; Numerous congenital malformations. Cyst communicated with scalp via bony canal.

Maier; 1861; *Virchows Arch. f. path. Anat. 20,* 536-541; 10 wk.; M; Teratoid apple.

Ogle, J.; 1865; *Brit. and For. Medico-Chirurg. Rev. 36:* 208-212; 22 yr.; M; Dermoids a) walnut b) ½ marble.

Heimpel; 1871; *Dissert. München;* 17 yr.; F; Dermoid.

Weigert; 1875; *Virchows Arch. f. path. Anat. 65,* 212-226; 14 yr.; M; Teratoma 3.5x3.5x3 cm.; No macrogenitosmia praecox, although tumor in pineal region.

Falkson; 1879; *Virchows Arch. f. path. Anat. 75;* 550-553; 16 yr.; M; Teratoid (?) 3x5x6 cm.; "Chondrocystosarcoma". No Macrogenitosomia praecox, although tumor in pineal region.

Irvine; 1879; *Tr. Path. Soc. London, 30:* 195-197, 7 yr.; F; Dermoid "large".

Beck; 1883; *Ztschr. f. Heilk, 4:* 393-410, 74 yr.; F; Teratoid 2.5x2.7x2.4 cm.; No hypophysis although tissue resembling anterior and posterior lobes within the tumor.

White; 1885; *Brit. Med. J. 1:* 282; "Boy"; M; Teratoid hazelnut; "Myoneuroma".

Sainsbury; 1886; *Tr. Path. Soc. London 37:* 57-59; Teratoid walnut.

Lannelongue and Achard; 1886; *Traité des kystes congenitaux,* Paris; 7½ yr.; F; Dermoid orange; Attached by stalk in depth of occipital bone.

Coats; 1887; *Tr. Path. Soc. London 38:* 44-52; 13 yr.; M; Teratoma 3x6.5x5 cm.; No rineal gland. No macrogenitosomia praecox "Adenoid chondrosarcoma". Tumor in pineal region.

Strassmann and Strecker; 1887; *Virchows Arch. f. path. Anat. 108:* 351-355; 3 yr.; M; Teratoid walnut; No clinical symptoms, incidental postmortem finding.

Scheuthauer; 1888; *Realencyclopadie der gesammten Heilkunde, von Eulenburg. Zweite Aufl. Wien, Leipzig, Urban and Schwarzenburg,* Bd. 15, P. 418; adult; Dermoid walnut.

Buzzi; 1888; *Mittheilungen aus der dermatologischen Klinik des Kgl. Charité-Krankenhauses zu Berlin,* Heft 5 p. 1; 22 yr.; M; Dermoid walnut.

Gauderer; 1889; *Inaug. Dissert., Giesen, C. von Münchow;* 12 yr.; M; Teratoma 2.5x3.5x2.5 cm.;

No macrogenitosomia praecox, although tumor in pineal region.

Kruse; 1891; *Deutsche. med. Wchnschr. 17:* 567-568; 28 yr.; M; Dermoid walnut; Incidental finding at autopsy.

Bonorden; 1892; *Beitr. z. path. Anat. u. z. allg. Path. 11:* 593-598; adult; M; Dermoids hazlenut; Incidental findings at postmortem.

de Lapersonne; 1893; *Arch. d'opht. 8:* 657-662; 18 yr.; M; Dermoid large nut; Complete removal in 2 stages one year apart. Cure.

Bathhurst; 1895; *Brit. Med. J. 1:* 1203-1204; 54 yr.; F; Dermoid 5 cm. diameter; Rupture into 3rd ventricle caused death.

Gutzeit; 1896; *Inaug. Dissert. Konigsberg. i. Pr., E. Erlatis;* 8 yr.; M; Teratoma 4x3.25 cm.; Genital precocity, (tumor in pineal region).

Saxer; 1896; *Beitr. z. path. Anat. u. z. allg. Path. 20:* 399-412; 7 wk.; F; Teratoma 8x10x12 cm.; Attached to tela chorioidea of 3rd ventricle.

Boestroem; 1897; *Centralbl. f. allg. Path. u. path. Anat. 8:* 1-98; 20 yr.; M; Dermoid 4.4x3.3 cm.

Lovett and Councilman; 1897; *J. Exp. Med. 2:* 427-438; 7 mo.; M; Teratoma 1320 g.; A less complex teratoma removed from right testicle at age three weeks.

Tannenhain; 1897; *Wien. klin. Wchnschr. 10:* 494-496; 25 yr.; M; Teratoid "large"; Extension of cystic contents into lateral ventricle; called "dermoid".

Eberth; 1898; *Virchows Arch. f. path. Anat. 153:* 71-74; 75 yr.; F; Teratoid split pea.

Trachtenberg; 1898; *Virchows Arch. f. path. Anat. 154: 274-291; 55* yr.; M; Dermoids small to 4 cm.

Ogle, C.; 1899; *Tr. Path. Soc. London. 50:* 4-12; 6 yr.; M; Teratoid 2.5 cm.; Macrogenitalia, hypertrichosis; tumor in pineal region.

Oestreich and Slawyk; 1899; *Virchows Arch. f. path. Anat. 157:* 475-484; 4 yr.; M; Teratoid small apple; Macrogenitalia, hypertrichosis, low-pitched voice, large size. No pineal gland., tumor in pineal region.

Benda; 1900; *Berlin. klin. Wchnschr. 37:* 1205-1210; 38 yr.; M; Teratoid walnut; "Teratoma" Hypophysis markedly atrophic.

Neumann; 1900; *Inaug. Dissert., Konigsberg i. Pr., J. Lowenthal;* 27 yr.; M; Teratoma 4x3x 2.5 cm.; Duration of symptoms only seven weeks.

Hlava; 1902; *Abstract in Centralbl. f. Chir. 29:* 905; 46 yr.; M; Teratoid; Called dermoid by author although said to have derivatives of all three germ layers.

Ruschhaupt; 1903; *Zentralbl. f. allg. Path. u. path. Anat. 14:* 945-952; 24 yr.; M; Dermoid hazelnut; More tumor in a defect in midline of occipital bone.

Raymond, Alquier and Courtellemont; 1904; *Rev. neurol. 12:* 635-636; 34 yr.; M; Dermoid nut; Probably rupture of main cyst.

Hulst; 1904; *Beitr. Geburtsh. 8:* 169-184; newborn: teratomas.

Buzzard, E. F.; 1904; *Tr. Path. Soc. London. 55:* 330-332; 14 yr.; M; Dermoid; Since symptoms began at puberty, (?) increase sebaceous secretion causing tumor to enlarge.

Erdheim, J.; 1904; *Sitzungsb. d. k. akad. d. Wissensch. Math-natur Kl. 113:* 537-726; 51 yr.; M; Teratoid 1x2.5 cm.; Cystic contents in subarachnoid space. Called dermoid by author.

Erdheim; 1904; 36 yr.; M; Dermoid 3cm. diameter.

Erdheim; 1904; 22 yr.; M; Dermoid 2x3x4 cm.; Incidental finding at postmortem.

Erdheim; 1904; 21 yr.; M; Dermoid 3x3.5x3 cm.; Incidental finding at postmortem.

Fromme; 1905; *München. med. Wchnschr. 52* (2); 1304; premature; Teratoma 640 g; Anlage of almost all organs of body.

Rows; 1906; *Rev. Neurol. and Psychiat. 4:* 338-344; 77 yr.; M; Dermoid 2.5 cm.

Rows; 1906; 73 yr.; M; Teratoid hen's egg; Convulsions for over thirty-eight years.

Ziehen; 1906; *Neurol. Zentralbl. 25:* 286. Dermoid.

Bregman and Steinhaus; 1907; *Virchows Arch. f. path. Anat. 188:* 360-393; 48 yr.; F; Teratoid 2 cm. diameter; Symptoms only four weeks.

Kon; 1908; *Beitr. z. path. Anat. u. z. allg. Path. 44:* 232-273; 37 yr.; M; Teratoma Hazelnut; Hypophysis separate from tumor in pituitary region.

Frankl-Hochwart; 1909; *Deutsche Ztschr. f. Nervenh. 37:* 455-465; 5 yr.; M; Teratoma 3x 5x2.5 cm.; Pineal tissue .2x.8 cm. still present; Macrogenitosomia praecox; Congenital defects —bronchial fissure. Hemangiomas. Tumor in pineal region.

Hecht; 1909; *J. A. M. A. 53:* 1001-1005; 11 yr.; f; Teratoid 2x2.5 cm.

Strohmeyer; 1910; *Beitr. z. path. Anat. u. z. allg. Path. 47:* 392-398; 40 yr.; F; Teratoid apple; symptoms only a few weeks.

Winteler; 1910; *Zentralbl. f. allg. Path. u. path. Anat. 21:* 668; M; Dermoid Hen's egg; Sudden onset of symptoms due to rupture of cyst.

Bailey and Jelliffe; 1911; *Arch. Int. Med. 8:* 851-880; 12 yr.; M; Teratoid 4 cm.; Obesity and increased growth but infantile voice and genitals, tumor in pineal region.

Schulgin; 1911; *Sowrem. Psichiat. Moscow. 1:* 143,—*Abstract in Zentralbl. f. allg. Path. u. path. Anat. 22:* 963; 51 yr.; M; Dermoid ruptured.

Kischensky and Tiesenhausen; 1912; *Abstract in Zentralbl. f. allg. Path. u. path. Anat. 23:* 358; 24 yr.; M; Teratoid pigeon's egg; Also a pea sized tumor at junction of falx cerebri and tentorium with neuroepithelial tubes, glia and nerve fibers.

Cushing; 1912; *The pituitary body and its disorders.* Philadelphia, J. B. Lippincott Co. p. 43 and p. 102; 16 yr.; F; Teratoid golf ball; Called Teratoma by Dr. Welch.

Cushing; 1912; 35 yr.; M; Teratoid 5 cm. diameter; Called mixed congenital tumor by Dr. Welch.

Kamber, S; 1912; *Inaug. Dissert. Berlin, Emil. Ebering;* 15 yr.; M; Teratoma small apple;

Pineal gland not included in tumor in that region.

Hymans, v.d., Bergh, v. Hasselt; 1913; Nederl. tijdschr. v. geneesk. 1: 1271-1287; 8 yr.; M; Teratoma marble; Macrogenitosomia praecox. Tumor in pineal region.

Hueter, 1913; *München med. Wchnschr. 60:* 895; 19 yr.; M; Teratoma pigeon's egg; no macrogenitosomia praecox. Histologic report by Berblinger. Tumor in pineal region.

Takeya; 1913; *Nisshu. Igaku. Vol. 3;* 10 yr.; M; Teratoma; Macrogenitosomia praecox, no histologic data. Tumor in pineal region.

Fukuo; 1914; *Inaug. Dissert., Munich, Kastner and Callwey;* 19 yr.; M; Teratoma walnut; Complete formation of wall of upper gastrointestinal tract. No macrogenitosomia praecox. Tumor in pineal region.

Kato; 1914; *Jahrb. f. Psychiat. 35:* 43-58; 16 yr.; F; Teratoid pigeon's egg.

Schmincke; 1914; *Munchen. med. Wchnschr. 61:* 2043-2044; 22 yr.; M; Teratoma walnut; No macrogenitosomia praecox. Tumor in pineal region.

Schmincke; 1914; 19 yr.; M; Teratoma walnut; No macrogenitosomia praecox. Tumor in pineal region.

Teutschlaender; 1914; *Virchows Arch. f. path. Anat. 218:* 224-248; 50 yr.; M; Dermoid 9.2x 8.2x7.7 cm.

Odermatt; 1915; *Inaug. Dissert. Zurich, Leeman and Company;* 10 yr.; M; Teratoma 3x2.4x2.7 cm.; macrogenitosomia praecox. Tumor in pineal region.

Burmeister; 1915; *Bull. Johns Hopkins Hosp. 26:* 410-412; 60 yr.; M; Teratoid 4.7x2.5x3.5 cm.; Called "chondro-fibro-epithelioma" and "teratoma".

Gautier; 1916; *Frankfurt. Ztschr. f. Path. 19:* 247-291; 9 mo.; F; Teratoid 1.3x1.1 cm.; Hypophysis normal except for extension through sphenoid bone into roof of pharynx.

Barron; 1916; *J. Cancer Research. 1:* 311-330; 19 yr.; M; Teratoma 4.2x3x4.4 cm.

Bosco; 1917; *Proc. Pan-Am. Sc. Cong. Washington. 10:* 522-528; 30 yr.; M; Teratoma 2x3x4.0 cm.

Sztanojevits; 1918; *Neurol. Centralbl. 37:* 784-786; 32 yr.; M; Teratoid fist.

Meyer; 1918; *Allg. Ztschr. f. Psychiat. 74:* 521-526; 11 yr.; M; Teratoma 4 cm.; Pineal gland not found. Increased growth, hypertrichosis. Tumor in pineal region.

Boehm; 1919; *Frankfurt. Ztschr. f. Path. 22:* 121-146; 9 yr.; M; Teratoma 5 cm.; Macrogenitosomia praecox. Tumor in pineal region.

Raueiser; 1919; *Klin Monatsbl. Augenh. 63:* 118-130; 16 yr.; F; Teratoid hen's egg; Operative removal. Cure.

Frank; 1920; *Berlin. Klin. Wchnschr. 57:* 888; 20 yr.; M; Teratoma 3.4x2.4x2 cm.; Symptoms only eight weeks.

Giebel; 1921; *Frankfurt. Ztschr. f. Path. 25:* 176-190; 15 yr.; M; Dermoid walnut; No trace of pineal gland. Tumor in pineal region.

Lohlein; 1921; *München. med. Wchnschr. 68:* 1036; Same case as Klapproth.

Luce; 1921; *Deutsche Ztschr. f. Nervenh. 68:* 187-210; 5 yr.; F; Teratoma large plum.

Klapproth; 1922; *Centralbl. f. allg. Path. u. path. Anat. 32:* 617-630; 15 yr.; M; Teratoma 4.5x3x 2.5 cm.; Infantile habitus, pineal adenoma with much mitosis, symptoms only six weeks.

Horrax; 1922; *Arch. Neurol. & Psychiat. 8:* 265-285; 23 yr.; M; Dermoid egg; Two operations recovery.

Horrax; 1922; 3½ yr.; F; Dermoid 5.5x4x3 cm.; Operation Death 2-3 months later.

Horrax; 1922; 2 yr.; F; Teratoid 2.4x1x.8 cm.; Operation Death 3½ months later.

Mendel and Unger; 1923; *Klin. Wchnschr. 2:* 1270; 32 yr.; M; Dermoid 200 cc.; Aspiration of cyst. Recovery.

Globus; 1923; *Arch. Neurol. and Psych. 9:* 417-430; 6 yr.; F; Teratoid 4x3.5 cm.; Hypophysis nearly normal but diabetes insipidus present.

Schuster, J.; 1925; *Schweiz. Arch. f. Neurol u. Psychiat. 16:* 327-331; 6 mos.; F; Dermoid "large".

Horrax and Bailey; 1925; *Arch. Neurol. and Psychiat. 13:* 439-441; 3 yr.; M; Teratoid 4x7 cm.; Macrogenitosomia praecox. Tumor in pineal region.

Helly; 1925; *Virchows Arch. f. path. Anat. 254:* 573-578; 36 yr.; F; Teratoma; Sebum and hair in subarachnoid space. Twenty years before "meningitis".

Cushing; 1925; 2 yr.; M; Dermoid; Bone defect in occiput in x-ray.

Rand; 1925; *Arch. Neurol. and Psychiat. 14:* 346-357; 40 yr.; F; Dermoid "large"; Attached to dura. Concentric shadow in x-ray.

McEachern; 1925; *Canad. M. A. J. 15:* 942-943; 33 yr.; M; Dermoid 11x5x7 cm.; Double ureter also present.

Derman; 1926; *Virchows Arch. f. path. Anat. 259:* 767-772; 1 mo.; Teratoid child's fist; "Teratoma biphyllicum cerebri".

Belloni; 1926; *Riv. di pat. nerv. 31:* 205-222; 42 yr.; M; Dermoid 10 cm.

Gorog; 1927; *Frankfurt. Ztschr. f. Path. 35:* 265-273; 50 yr.; F; Dermoid hazelnut; Attached to choroid plexus in right lateral recess of fourth ventricle.

Kopriwa; 1927; *Med. Klin. 23:* 645-646; 30 yr.; F; Dermoid 4x5 cm.; Brain tumor an incidental finding. Horsheshoe kidney; fusion of third and fourth ribs.

Todesco; 1927; *Lancet. 2:* 1340-1341; 21 mo.; F; Dermoid Hen's egg; Small hairy cyst over external occipital protuberance also.

Horrax and Bailey; 1928; *Arch. Neurol. and Psychiat. 19:* 394-413; 9 yr.; M; Teratoid; Slight pubertas praecox. Tumor in pineal region.

Hesse; 1928; *Zentralbl. f. allg. Path. path. Anat. 42:* 555; 1½ yr.; Dermoid hazelnut.

Downes; 1929; *J. Coll. Surg. Australia. 2:* 268-270; 7 yr.; F; Dermoid 4.6 cm.; Death following operation.

Kraus; 1929; *Virchows Arch. f. path. Anat. 271:* 546-555; premature; F; Teratoid 3.7x3 cm.; Dumbbell-shaped tumor in pituitary region and in pharynx connected through open craniopharyngeal canal.

Lua; 1929; *Deutsche Ztschr. f. Nervenh. 109:* 212-230; 25 yr.; M; Dermoids a) 7x7.5x6 cm.; b) plum c) 1 mm.; Ruptured into right ventricle; fat scattered in subarachnoid space.

Verebély; 1929; *Abstract in Zentralbl. f. Ophth. 20:* 130; 20 yr.; F; Dermoid; Radical removal and cure.

Altman; 1930; *Wien klin. Wchnschr. 43:* 108-111; 11 yr.; M; Teratoid 4x4.5x5.5 cm.; Obesity. No macrogenitosomia praecox; Much pineal tissue still present despite large tumor in pineal region.

Hosoi; 1930; *Arch. Path. 9:* 1207-1219; 27 yr.; M; Teratoma 3x3x3.5 cm.; Dwarfism, kyphosis, scoliosis associated.

Kornfeld; 1930; *Virchows Arch. f. path. Anat. 278:* 165-177; 33 yr.; M; Teratoid; Symptoms 15 yrs.; Hair extended into cerebellum.

Kwan; 1930; *Arch. Neurol. Psychiat. 24:* 1292-1293; 19 yr.; F; Dermoid 5 cm.; Tumor extended through defect in occipital bone.

Sarvaas; 1930; *Frankfurt. Ztschr. f. Path. 40:* 210-223; 12 yr.; M; Teratoma 3x6.5x12 cm.; Tumor attached to hypophyseal stalk. Growth of tumor out of nose during life.

Brock and Klenke; 1931; *Bull. Neurol. Inst. New York. 1:* 328-342; 27 yr.; M; Teratoid 50 cc.; Crescentric shadow in x-ray; successful removal. Recovery.

Chasan; 1931; *Schweiz. Arch. f. Neurol. u. Psychiat. 27:* 64-107; 2 days; M; Teratoma; Malformation and teratoma involving most of brain.

Shapiro; 1931; *Arch. Path. 11:* 22-43; 36 yr.; F; Teratoid 2.5x2.5x2 cm.; Polyuria without involvement of pituitary gland.

Rigby; 1932; *Brit. J. Radiol. 5:* 349-350; Same case as Scott, 1932.

Scott; 1932; *Proc. Roy. Soc. Med. 25:* 720-721; 2½ yr.; M; Teratoid; No operation or autopsy to confirm x-ray diagnosis.

Bauditz; 1933; *Ztschr. f. d. ges. Neurol. u. Psychiat. 144:* 135-147; 48 yr.; M; Dermoid; Ruptured, Cystic contents along entire base of brain.

Heymann; 1933; *Zentralbl. f. Chir. 60:* 842; 45 yr.; M; Dermoid; Large calcified shadow in x-ray; Removal with cure.

Mari; 1933; *Riv. di pat. nerv. 41:* 531-545; 8 yr.; Teratoid 11x7.5x6.9 cm.

Schuster, H.; 1933; *Zentralb. f. allg. Path. u. path. Anat. 59:* 163-165; 1 day; Teratoma.

David, Loisel, Corria, and Brun; 1934; *Rev. Neurol. 2:* 79-86; 19 yr.; F; (?) Teratoid orange; Tumor type virtually unique, containing cellular monstrosities of many glial types.

Baxter and Haber; 1934; *J. Pediat. 4:* 795-797; 12 yr.; M; Dermoid 60 gr.

Phillips and Stone; 1934; *Bristol. Med-Chir. J. 51:* 247-252; 18 yr.; M; Dermoid 5x6 cm.; Cerebral substance not invaded.

Ferraris; 1935; *Ann di laring, otol. 35 (10 new series) 1:* 23-30; 14 yr.; F; Teratoma small egg; No microscopic description.

McLean; 1935; *Surg. Gynec. and Obst. 61:* 523-533; 6 yr.; M; Teratoma 2x2x2.5 cm.; Pineal gland not involved, no sexual precocity. Tumor in pineal region.

Harding and Naish; 1935; *Lancet. 1:* 77-80; 9 yr.; F; Teratoid 3x3.5x4.5 cm.; Clinically lack of expression of emotions.

Harding and Naish; 1935; 1 yr.; F; Teratoma 3x 5.5 cm.

Manca; 1935; *Arch. ital di anat. e istol. pat. 6:* 371-394; 20 yr.; M; Teratoma over 150 gram; Pineal gland not involved. Tumor in pineal region.

Scheinker; 1936; *Deutsche Ztschr. f. Nervenh. 140:* 217-228; 40 yr.; M; Dermoid plum size; First symptoms only ten days before death; rupture of cyst into ventricle causing ependymitis and occlusion of aqueduct.

Toropova; 1936; *Sovet. psikhonevrol. No. 10:* 65-70; 46 yr.; F; Teratoid; Arnold-Chiari deformity of cerebellum.

Malykhova; 1936; *Sovet. Khir. No. 12* p. 998; 26 yr.; M; Dermoid.

Love and Kernohan; 1936; *J. A. M. A. 107:* 1876-1883; 30 yr.; M; Dermoid; Surgical removal with cure.

Love and Kernohan; 1936; 2½ yr.; Dermoid; Death eleven days postoperatively.

Love and Kernohan; 1936; 30 yr.; M; Dermoid; Death five days postoperatively.

Love and Kernohan; 1936; 30 yr.; M; Dermoid; Grand mal seizures sixteen years.

Alpers; 1937; *Arch. Neurol. and Psychiat. 38:* 291-303; 39 yr.; M; Teratoma 2x2.5x3.5 cm.; Case used for study of disorder of personality in hypothalamic tumor.

Farthing and Love; 1937; *Proc. Staff Meet., Mayo Clin. 12:* 721-724; 24 yr.; F; Dermoid 140 gm.; Symptoms over twenty years. Complete removal with cure.

Gladstone; 1937; *J. Indiana M. A. 30:* 282-284; 1 yr.; F; Dermoid 2 cm. diameter; Defect in midline of occipital bone. Secondary infection via tract through this defect.

Kahn; 1937; *Arch. Neurol. and Psychiat. 38:* 833-842; 9 yr.; M; Teratoma 2 cm. diameter; Complete removal with cure. Pineal tissue in tumor of pineal region.

Jensen; 1937; *Acta ophth. 15:* 193-203; 13 yr.; F; Teratoid 3.5x4.5x5.5 cm.; Hypophysis could not be found.

Miller; 1937; *Australian and New Zealand J. Surg. 7:* 74-77; 11 yr.; F; Dermoid 60 gm.; Complete surgical removal with cure.

Munro and Wegner; 1937; *New England J. Med. 216:* 273-279; 26 yr.; F; Dermoid 4x6 cm.; Tumor almost completely removed, recovery.

Stender; 1937; *Zentralbl. f. Neurochir. 2:* 114-122; 36 yr.; F; Dermoid mandarin; Complete removal with cure.

Stender; 1937; 60 yr.; F; Dermoid man's fist; Removal with cure.

Bochner and Scharff; 1938; *Arch. Surg. 36:* 303-329; 9 yr.; M; Teratoma 5.5x3.5x5.5 cm.

Verbiest and Zeldenrust; 1938; *Nervenarzt. 11:* 366-369; 17 yr.; M; Teratoid; Death following operation.

Weber; 1939; *Zentralbl. f. Neurochir. 4:* 47-57; 41 yr.; F; Teratoid small fist; Successful operative removal.

Weber; 1939; 25 yr.; F; Teratoid mandarin; Successful operative removal "teratoid cyst—mixed tumor containing connective tissue".

Weber; 1939; 2 yr.; F; Teratoid apple; Successful operative removal.

Weber; 1939; 5 yr.; M; Teratoma walnut; Operative removal.

Weber; 1939; 13 yr.; M; Teratoid potato; Death following operative removal.

Weber; 1939; 12 yr.; M; Teratoma apple; Operative removal.

Weber; 1939; 18 yr.; M; Teratoid walnut; Death following operative removal.

Bailey, Bucy, Buchanan; 1939; *Intracranial Tumors of Infancy and Childhood,* Chicago, University of Chicago Press, 598 pp.; 13 yr.; M; Teratoid 6x3x3.5 cm.; Macrogenitosomia praecox. Tumor in pineal region.

Bailey, Buchanan, Bucy; 1939; 6 mo.; F; Teratoma 10.5x7 cm.

Sweet; 1940; *Arch. Neurol. and Psychiat.* (In Press); 54 yr.; F; Teratoid 7 cm.; Remission in symptoms for eight years due to a spontaneous third ventriculostium.

Sweet; 1940; 45 yr.; F; Dermoid 30 gms.; Operative removal with cure.

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Obesity as a Manifestation of Personality Disturbance

THOMAS A. C. RENNIE, M.D.
Baltimore, Maryland

Dr. Rennie was graduated from the Harvard Medical School in 1928. He is an associate professor of psychiatry and neurology at the Johns Hopkins University School of Medicine and he has written a number of papers which have appeared in the leading medical journals.

Tradition has characterized the obese individual as cheerful and contented, and we find that belief expressed in axioms such as, "Laugh and grow fat." The physician managing obesity, however, not infrequently finds the very opposite, and one sees certain individuals turning more and more toward food under the worries and stress of life. In the management of these obesities, one may have to deal with fundamental psychiatric disturbances: states of tension, anxiety, and depression, which sometimes explain the development of the obesity and offer a more constructive mode of therapeutic attack. The purpose of this paper is to describe a series of such patients in whom psychiatric investigation revealed life factors responsible for the increasing weight.

Obesity is a problem that is exceedingly complex and takes into consideration a wide variety of factors, although investigators have paid scant attention to the possibility that personality may play an important, if not leading role, in the disorder. Neglect of this in the control of obesity may lead to failure of therapeutic effort.

The physician's first concern, naturally, is for the elucidation of the fundamental physiological disturbance. With this in mind, it may be well to state the variety of funda-

mental disturbances that must always be considered and excluded. Congenital factors may be concerned and family history needs evaluation. Abnormal regulative hypothalamic centers may be disturbed. Disturbances in appetite and demand for excessive food and drink, dislike of physical exercise, or abnormal fatigability may be present with or without the many endocrine disturbances which may be active. Among the endocrine possibilities one must always rule out lowered basal metabolism or other specific dynamic action of foods. Barker[1] has summarized the possible disturbances, and enumerates the obesity developing after removal of the ovaries or after castration, the obesities of hyperinsulinism, hypoglycemia, occasional carcinomas of the pancreas, hypogonadal obesity, hyperfunction of the suprarenal cortex, adoposis dolorosa. The hypophysis may play its role in adoposogenitalis, and certain obesities of neural origin may give rise to fatness of the lower half of the body (lipodystrophia) and fatness of one lateral half of the body.

The great majority of obese patients, however, never show manifestations of these relatively rare conditions. Hochman[2] points out that in 400 consecutive cases of his own these obscure factors have been present relatively rarely and that in most cases the problem reduces itself to the fact that more food is consumed than is necessary for energy requirements. In the discussion of these dietary factors he feels it necessary to point out the role of psychological factors in the production of tensions and conflicts responsible for overeating. While granting that a certain predisposition is necessary, he finds that compulsive eating occurs as a relief from fear, as a reaction against anxiety, and he points out the wisdom of tackling the psychological background as well as drug and caloric supervision. His review of the literature makes it unnecessary to quote further investigators at this point, other than to mention specifically Myerson's studies in autonomic pharmacology, in one of which[3] he defines a disturbance of appetite-regulating mechanism for which he uses the term "anhedonia". He lays stress on the social habits governing the intake of food and describes a state of loss of general satisfactions leading to a restless seeking for stimulation which may find its outlet in frequent eating. Thus he concludes that this syndrome of anhedonia may be associated with neurosis and urges the use of psychotherapy in addition to any specific medication such as the use of benzedrine sulfate.

While it is known that in most cases of mood disturbance, such as depression, the tendency is for loss of weight, there are a few such individuals in whom the opposite occurs. In our own cases to be described it will be seen that personality factors productive of states of tension, depression, anxiety, and sometimes hypoglycemia attacks may result in a considerable increase in weight during the period of personality upset. In other cases the obesity is definitely tied up with the psychoneurotic picture serving a specific function in the total psychoneurotic disturbance. Uncooperativeness, inability to remain strictly on diet, lack of fundamental desire to reduce, may all contribute to the physician's therapeutic task and may be not at all evidences of conscious lack of cooperation, but manifestations of upsets of which the patient himself may have little or no awareness.

The first case shows clearly the complex personality factors which are interwoven with the obesity problem. In this case we see clearly how eating serves as a solace in times of unhappiness, and obesity plays the dominant role in the whole life activities, having to do wth the selection of a profession, complicating the family and social adjustments, constituting, in fact, the main pivot around which the entire life development and adjustment is best understood.

Case 1—The patient was a 21-year old unmarried dietitian weighing 234 pounds, who entered the medical service 10/17/38, complaining of obesity, headache and fatigue. She earnestly hoped that some endocrine factor would be found which would make her obesity respectable and simple. Thorough physical examination revealed a generalized obesity of 234 pounds. General physical examination was otherwise negative with a B.M.R. of −4, x-ray of skull normal, menstrual irregularity associated with the obesity, but no definite evidence of endocrine abnormality. Placed on a 1,000-calory diet she failed to lose weight, showed acetone only once, may have been cheating with her food

intake.

To the psychiatrist the girl launched into a long story of her unhappy life, during which she cried most of the time in a decidedly childish, self-pitying manner. What she told was probably true but had to be weighed in the light of the parents' anxiety. She was an only child, although she had an adopted older sister who was actually a first cousin. Early in life, according to her, her mother, her aunt, and her grandfather began hounding her about her obesity, calling her a monstrosity fit for a circus, saying she ate like a horse. She had been called bullheaded, stubborn, weak, lacking in will-power. Her father was always sympathetic, with the result that she gravitated to him and grew further and further away from her mother. This brought out a situation of resentment between her parents, leading to frequent quarrels. She felt that the family was ashamed of her. During the years away at college, she gained a little self-confidence and got on better. She had, however, never had dates or any attention from men. The patient claimed that periodically she reacted to the home situation with resentment and anger—"to go out and eat was an outlet for it. I'm so bitter and I don't want to be bitter. I have a most horrible resentment against my mother." She spent one protracted period at Battle Creek where she lost forty pounds, but was caught eating on the side. The same thing happened at Hot Springs. In other words, this girl had never been able to discipline herself enough to stay on a diet. Her neurotic resentment was a large factor in her eating bouts. She said quite frankly, "I get a craving for candy, ice cream, cookies, and homemade cake, and I simply cannot resist it. I have cheated many, many times. They are always suspecting me of eating so I said to myself, 'Oh hell, why not?', and then I would eat."

According to the patient, the mother, aged 47, had gone through the menopause and had "insane ideas that people are against her." Her grandfather had an ungovernable temper. As a child, the patient was nervous, high-strung, talked in her sleep, suffered with malnutrition. She showed neurotic traits of nightmares, nail-biting, enuresis up to the time of examination (interestingly, only when at home, never when visiting), and temper tantrums. She soon learned, however, that her temper availed her nothing and learned to concede to every argument. She finished high school at 15. She wanted to study endocrinology but decided on dietetics. At college her supervisor refused to get her a job, saying she was so fat she was hardly a good representative of dietetics. Sex life consisted in an abortive love affair at 15 with an enlisted man in the army who paid her some mild attention. She had not heard from him for years. In temperament she claimed to be quite religious and was interested in mysticism, metaphysics and palmistry.

The mother gave a picture of an extremely troubled household revolving around this girl's obesity. The maternal side were all overweight, ranging from 250 pounds upward. The mother stated frankly that she felt the girl showed weakness of attitude and character and it was evident that her desire to have her daughter beautiful and attractive had made of her a martinet who quite frequently called the patient a monstrosity, who refused to let her have dates, and who was ashamed to have her seen by her friends. For years the mother alternated between praise, indulgence, coercion, and force. She complained that the girl was uncooperative, dishonest, lacking in pride and vanity, and "my child is an absolute stranger to me." The father, on the other hand, felt that the girl's life had been made utterly miserable by the mother's nagging and a very strained, uncomfortable relationship had developed between the parents.

This obesity problem, therefore, served a variety of functions for the patient. She had made herself the center of family attention; she had succeeded in estranging her parents. Her eating could roughly be compared with the urge of an alcoholic for alcohol in that it served for her a definite function in which she shifted the responsibility upon her mother.

The mother was encouraged to abandon her policy of overconcern and overemphasis which had only served to defeat its own purpose. She was asked in the future to make no further mention of the weight problem and to leave the responsibility entirely up to her daughter.

We see in this case a complex neurotic personality present from earliest childhood with its multiple neurotic traits, serving its function in keeping her the central target of

family solicitude and strife, with the final evolution of a temperament marked by bitterness and resentment, reacting to the accusation of eating by deliberately doing so. It seems difficult to avoid a conclusion that eating with this girl represents the expression of her maladjustment, much as the psychoneurotic would express it in other forms. Since it fills such a central role in her multiple difficulties, it seems that the prognosis for weight reduction is likely to be poor until the girl has been helped to a better understanding of her own complex make-up. As might have been expected, she wrote one year later that she had gradually slid away from a strict regime and her weight had increased to 240, where it still is. During her mother's intercurrent attack of kidney stones, the patient took over the management of the house, only to find herself resenting her mother's recovery and return to the management of the home. She still spoke of the tension in the home, her desire to get away from it. She twice consulted a psychiatrist at home, feeling that she gained something in a fuller understanding of the points of tension but stated, "I do watch what I eat, but tasting does go on."

The next case shows equally clearly the extent to which personality comes into the obesity problem. This man states frankly that eating is his solace in times of stress, saying that he could reduce at any time with adequate effort, and complaining that when he is worried, hunger comes over him like a madman.

Case 2—The patient was a 30-year old unmarried man weighing 417 pounds, referred by the medical department because of a gain of 110 pounds in weight during the last two years. In addition, he gave a history of drinking 3-4 gallons of water in twenty-four hours and of having nocturia three times nightly. He was apparently perfectly normal in health and size until the age of 18. At that time he broke his leg in an automobile accident and was laid up in bed in a cast for three months. During this three months he ate excessively, gaining from his normal weight of 168 to 198, saying it was his only solace. He seems to have acquired the habit of colossal eating then because there was nothing better to do. He spoke of being quite nervous because he had been told his leg would never be good enough to walk on, and "the more nervous I got from worrying about that, the more I would eat." Since that date he had never been able to break the habit of eating and, especially when nervous, developed a craving to fill his stomach. He confessed that he could diet "until something goes wrong and gets me nervous; then I've got to eat. It may be my nerves which are out of order." During extensive studies on the metabolism ward two years previously he reduced weight normally and appeared calm and untroubled. At that time he showed no polydipsia or polyuria.

Physical examination was essentially normal with no evidence of neurological or endocrinological involvement. The family history of obesity is striking. The neurological consultation and the ophthalmological examination of the visual fields revealed no basis for diagnosis of diabetes insipidus.

To the psychiatrist he spoke of yearning for a simple medicine which would make reducing easy. He admitted that on a diet he could reduce, but that the effort was too much for him. He said that he ate every time he felt hungry, which was four to six times a day, or when he was idle and had nothing to do. He would finish a meal and when his brother came in an hour later, sit down and have another full meal with him. He stated that when he was worried he ate and that hunger came over him "like a madman."

He was born in 1908, a small baby because his mother had typhoid during her pregnancy. He came of a family of heavyweights, although his 417 pounds topped the list. One brother was an epileptic. As a child, the patient was a troublemaker, breaking windows, etc. He roamed the streets selling newspapers until 3 a. m. He was once in Juvenile Court. Up to the time of examination he still screamed, argued and fought in his sleep. He periodically had a rash on his buttocks. He finished the eighth grade, just getting by. He had never had any consistent job, but helped out in his mother's store. He said frankly that the financial accounts were in horrible shape and he kept lying to her about the success of the business. His one chief recreation was a Turkish bath on Saturday nights. His sex life was free and easy. He picked up prostitutes on the street or his friends brought them to his home. Inter-

course occurred at least once a week. He suffered from premature ejaculation.

He claimed his life was miserable because of his fat. He could not get clothes to fit him. He worried about his epileptic brother who tried to boss him. He felt he was conspicuous, as he most assuredly was. He thought people were always teasing him about his weight—"I don't have no willpower—if I just had will-power. Can't you give me something to make me sick at my stomach and break me down?" He liked to play the lotteries and "I want to beat it so bad it aggravates me. I just live always in aggravation. The more I try to lie, the more I seem to get caught."

We deal in this case with a man of limited intelligence, shiftless habits, social irresponsibility, clearly of the type of psychopathic personality. He yearns for a medicine which will destroy his appetite, but frankly admits complete lack of will-power and lays the blame for his overeating on his nervousness, worry, aggravations. With such a personality make-up the outlook for any success in a reduction program is obviously poor. He has finally drifted from attendance at the endocrine clinic. While being studied on the metabolism ward and free of any particular worries, he was able to reduce weight normally so that we have every reason to assume that the problem is primarily related to the elements of his own personality which make him, when the least thing goes wrong, "go out and tear off huge quantities of food," whereupon his nervous upset is promptly quieted.

In the preceding two cases, family history of obesity is striking. In the next case there is a tendency to stoutness on the maternal side, but the familial component is less evident and the situational factors and psychiatric status are more clearly etiological.

Case 3—The patient is a 43-year old unmarried woman weighing 196 pounds who entered the medical service for two reasons: (1) to get a rest from the trying situation of caring for a senile father at home; (2) to get help in the reduction of her obesity. Physical examination revealed moderately even distribution of excess fat, weight 196, but otherwise completely negative examination, including B.M.R. of −3 and normal glucose tolerance curve. On a 1,000-calory diet she began losing weight promptly, losing 4¼ pounds in four days.

To the psychiatrist the patient stated frankly that she was tense and unhappy. Concerning this latter she stated that she had gained weight recently during the tension at home. Her father liked to eat frequently and she found that she did the same so that her caloric intake had actually increased considerably. However, she stated that when she was feeling down or tense, she got considerable lift from a chocolate soda. She recognized that she was tense and noticed her symptoms in afternoon fatigue, tightness of the muscles of the neck, occasional feelings of dizziness relieved by sugar or a sandwich —"sweets lift me like alcohol and give me the same pep as tea." All her life she had been subject to afternoon fatigue unless she was engaged in sports when she was unaware of it, but usually felt profoundly exhausted after the game was over.

She described this episode as similar to one nine years ago when her mother died, which was quite a shock to her. She felt the same tenseness in the back of her neck. At that time she gained weight due to increased eating. On a regime of massage and diet she recovered in about three months.

The immediate situation was that her father had become increasingly difficult to manage at home. Within the previous month there was a period of two weeks when she got no sleep at night because of her father's demands. Two weeks previously she had secured a night nurse to take over the burden and had slept reasonably well since. She had been constantly with her senile father, who got confused at night, demanded to go to his office. He was restless and indecisive, peremptory and demanding. She felt it was "getting" her and decided she needed a rest and a change. She had not been particularly depressed. She denied being restless but was a little irritable and tended to cry easily.

As a child she stated she was calm and placid; there were no neurotic traits. She had occasionally had hives. At college, with late hours, long study, and poor food, she became rundown, her menses stopped and her family physician took her out of school in the middle of the first year. With bed rest she recovered in four or five months. The second outstanding upset came after her mother's death. The

present illness represented the third well-defined period. In addition to the situation with her father, she had had some financial concerns, having had to take over the management of the family estate, and she was not in a position to afford adequate nursing help at home. She realized that the time would soon come when her father would have to be hospitalized, but dreaded the expense and the attitude of her father's friends who had no understanding of the burden he constituted. She was somewhat resentful of her sister who had evaded all responsibility about her father until the previous year when she returned home and offered help without doing anything about it. The patient felt that the sister had an eye on the father's money. Patient denied any sexual worries. She was a person of wide interests and talents, including painting, piano, dancing, skating, golf, reading, sewing, travel, etc.

All her life she was terribly shy although she had an exterior which belied it. Any new situation was difficult for her and she became soaked in perspiration. She was unusually sensitive, her feelings were easily hurt, she disliked criticism, tended to be suspicious. She was far too conscientious and leaned heavily upon the good-will of others. She naturally liked to eat and had enjoyed sweets all her life.

There seemed little doubt but that the patient was in a state of definite tension. The situation at home was more than she could bear with equanimity. In this situation of tension the old craving for food recurred and she began gaining weight. It is of interest to note her complaints of afternoon fatigue, dizziness, tightness of the muscles of the neck, relieved by taking carbohydrate. This type of fatigue would appear to be clearly tension in origin since glucose tolerance curve gave no reason to suspect hypoglycemia. She was recommended to make modifications at home and to get skilled help in the handling of her father. Hospitalization of him was discussed and recommended. She had a striking lack of understanding of her own personality make-up and readily and gratefully accepted the suggestions for the modification of her overconscientiousness, continuance and utilization of her assets and recreational outlets. She was advised to utilize continuous baths, systematic exercise, massage and occasional trips away from home for the relief of the tension.

In the next case the overeating seems definitely related to depression since the patient so specifically states that moodiness, loneliness and depression always resulted in her wanting to eat.

Case 4—The patient was a 37-year old married business woman who entered the medical service 6/26/39 for reduction of weight of 213 pounds, giving a history of consistent weight gain of ten pounds a year over a period of a number of years. She had a family history of obesity. There had been a hysterectomy ten years previously for ovarian cyst, but she had no menopausal symptoms following this. As a child she was plump, but in high school, following onset of menses, lost weight without dietary effort. Promptly following her operation she began to have a greatly increased appetite (although no menopausal symptoms), used to eat between meals and gained 30 pounds in the first year post-operatively. She had dieted off and on numerous times and was able to lose weight satisfactorily. Physical examination revealed nothing aside from the obesity and it was felt that no endocrine factor played a role.

In the account of the weight gain she stated specifically that whenever she was depressed she wanted to eat. Psychiatric examination revealed an interesting and complex background, which undoubtedly contributed the major share in her weight gain. The year following her operation she began helping her husband actively in his business, taking buying trips to New York. In New York she first noticed her increasing appetite and attributed it to loneliness. Whenever she was alone she tended to become moody and invariably got hungry. She was always likely to eat excessively under emotional situations. For example, three years ago, while both parents were ill and in hospital, the patient felt under considerable strain and would invariably eat just before going to the hospital to visit them. Following a night of poor sleep she always got up wanting to eat. Similarly she was hungry if she was dissatisfied with her accomplishment, left a task unfinished, or had a bad day at the store. She thoroughly disliked fat people, but said she was able to do anything except stop eating.

The background is that of a Russian-born

individual of an unhappy family atmosphere. The father was a rigid, strict, narrow person and the patient took the brunt of it. Always, she looked forward to getting out of the home. She was terrified of her father. As a child she was unattractive and plump and was always being compared unfavorably to a beautiful younger sister. She was a moody child who had occasional nightmares. She was an honor student through high school, but had to fight her father to get her education. She went to work in her father's store, which she disliked, had long hours, little recreation, was inadequately paid. Menstruation came as a terrific shock since she was totally unprepared for it, and it was accompanied by dysmenorrhea. She was given no sex instruction and was rather prudish about sex topics. The family objected to her going out with boys. Following her first kiss at 18 she thought she would be pregnant. Her father forbade her marriage to a young man without financial resources. She insisted on marrying him even though she was shocked when he told her that he had had sex relations. She wanted to break her engagement, but thought she should go through with it. The marital sex relations were an additional shock to her. She found her husband repulsive and thought sex relations were abnormal. She hated intercourse, but became reconciled to it after the birth of her first child. Years later a better adjustment was achieved, but her marriage has not been a very happy one. She early decided that since her marriage was such a disappointment she would make it a financial success. She put all her time into their store, living a narrow, unsatisfying life. Eight years ago she felt much attracted to another man, but her high standards of marriage would not permit her to enter into any relationship with him.

These years of dissatisfaction undoubtedly contributed a great deal to the episodes of moodiness and depression which in turn gave rise to the great desire for food, and seem to have been the major factors behind the excessive weight gain.

Psychiatric therapy was undertaken in conjunction with the dietary regime. She continued to lose weight slowly, but steadily. At the end of three months she went on an eating binge which followed an altercation with her husband over a necessary change in the personnel of the store. She said nothing about her resentment for several days and then turned to almost constant eating of bread and sweets. She finally demanded a conference with her husband and solution of the problem. After she had settled the issue she went back on her diet, saying spontaneously that the whole incident had taught her that it was not a question of physical hunger but a problem of emotional upset which had led to her eating. Three weeks later she came back into the hospital following another weekend of lapsing from her diet. This time she recognized it as the culmination of the week of tension and fatigue preceding it, which in turn had been definitely stirred up by the realization that she had provided little family life for her son to whom she was devoted. The analysis of the two periods of failure, therefore, clearly indicated the extent to which worry and resentment were the motivating factors behind her dietary lapses.

In this previous case we deal with an obese individual from an obese family. While the initial gain in weight seemed to follow hysterectomy, she herself quite clearly relates the weight gain to increased food consumption, which she in turn relates specifically to episodes of discouragement and depression.

In the next case we deal with a woman with definite depression with concentration difficulties. She has a story of years of difficulties, repeated operations, divorce, worry and insecurity, and the gradual centering of her own attention more and more on her specific symptomatology.

Case 5—The patient was a 43-year old divorced, employed woman who entered the medical service 4/11/37 for the purpose of reducing her weight of 223 pounds prior to submitting to operative repair of a ventral hernia. She stated that she was last well four years ago and complained of fatigue on awakening, dizzy attacks, nervousness, crying spells, abdominal pain prior to menses, migraine headaches, abdominal hernia, constipation, fear that her mind was being affected because of poor memory; spontaneous crying spells without adequate reasons, tenseness, inability to relax. Physical examination revealed generalized and symmetrical obesity. There were no indications of hyperthyroidism; B.M.R. +6. X-ray of the skull showed normal sella turcica. B.P. 105/70. All other

routine examinations were negative aside from a typical neurotic excoriation of the skin, arms, shoulder, back and legs. She ascribed most of her symptoms to worry, fear that her mind was affected, inability to remember, lack of ambition. Interestingly, the complaint that brought her to hospital was definitely secondary for, as she said, "I complained most of the hernia because this other thing that I feel is unreal and I couldn't explain it, so when I go to doctors I talk about the hernia which really bothers me some, but it is in the hopes that they'll do something about my tired, worried feelings."

To the psychiatrist she appeared extremely overtalkative, reciting her story with great attention to minor details. She stated she had always been a nervous person, but was much worse following an abdominal operation in 1933 for removal of both ovaries, uterus, tubes and appendix. Since then she had suffered from excessive fatigue; her work had become harder and harder for her to accomplish. She worried when unable to keep up with her usual pace. She had a constant fear that she would not get her work done properly. She developed itchiness of the skin and scratched it until it bled. For this condition she consulted sixteen doctors, all of whom attributed it to nerves. She noticed her concentration was poorer, her memory failing. She had an increasing intolerance of noise so that the rattling of paper made her very tense. Within the past year she had dizzy spells and heaviness in the head. She had lost interest in her personal appearance, spent most of her time in bed, felt she had little to live for. Sleep was poor, but her appetite had remained good throughout the entire period. She stated her spirits were blue.

She had a normal childhood without neurotic traits. Her mother died when she was 7. From then on she lived with various relatives wherever they needed someone to help with housework or care for children. Her schooling was irregular and she went through only the seventh grade. She married at 19 a man she did not love because she was anxious to get away from the life of drudgery she was leading. He drank heavily and was constantly losing jobs because of his alcoholism. Her life with him was one of constant moving to new jobs, none of which he could keep. Later he took to narcotics and other women, staying away from home for days or weeks. Her life was one of constant uncertainty and terror as he had a violent temper and once shot at her when he was drunk. Ten years ago, in desperation, she left him, and four years ago divorced him. During the six years before her divorce he would send for her when stranded and she would help him out. She obtained a job for herself at $75.00 a month. She had one daughter who was working. Her own advancing age and the threat of her daughter's impending marriage seemed to contribute further insecurity.

It was in this setting of years of insecurity that she began putting on weight and it was two months following her divorce and following pelvic operation that she began rapidly to accumulate excess weight. In the hospital she stated she felt a great deal more secure and within six days had begun satisfactorily to lose weight on a 1,000-calory diet.

In this case also we deal with a definite depression characterized by crying spells, concentration difficulties, fear of insanity, tension, and a variety of consequent symptoms including vertigo, morning fatigue, tremulousness, constipation, etc. She has a lifelong history of migraine, neurasthenic exhaustion, tension, all aggravated by the complex circumstances of her life. One wonders how many of the operations were really indicated. The final medical diagnosis was ventral hernia, obesity, chronic tonsillitis, and psychoneurosis, the recommendation being that treatment be entirely psychiatric.

In the next case the patient entered the hospital complaining of obesity and of fatigue of 8 years' duration. She is an individual who was quite well until the disruption of her usual mode of life, consisting in outdoor exercise and social activities. She immediately began to gain weight, gaining from 125 to 190 pounds. The setting in which this developed gives ample indication of the personality disturbance providing the setting for the weight gain.

Case 6—The patient was a 31-year old married woman who entered the medical service for weight reduction, having gained from 125 to 190 pounds during the eight years of her marriage. The medical examination revealed essentially negative findings. All laboratory tests were within normal limits with the exception of the glucose tolerance curve of 103,

152, 170, 98, and 54 at the end of three hours. She spoke of having an enormous appetite and had a family history of obesity.

The psychiatric consultant obtained a history of increasing nervousness in association with fatigue, tremulousness, poor sleep, irritability, ups and downs of mood, poor concentration and memory, and frequent ideas that she might be going crazy.

The events of her life were very revealing. An 11-year law suit in which she was involved for a bankrupt estate contributed a great deal. A break with her mother and family had led to many embarrassing events. Her childlessness had been a great disappointment. Her husband had had no sexual urge for many months; the patient herself had marked drive which was left unsatisfied. She was sensitive and wanted to avoid hurting her husband's feelings.

Her past life was somewhat disjointed. She was in nine different schools, never being a good student in any. Her parents separated because of her mother's interest in a younger man. The patient was devoted to her father and immediately joined him in the ensuing battle. His death was a profound shock to her. She had a number of hobbies. She and her husband retreated to a mountain cabin as often as possible, where she got some relaxation. Her husband was a Catholic; she, Protestant. She would not want her children raised as Catholics, but his family would force such training.

One notes in this case also the complaint of fatigue which seems definitely of a neurasthenic variety. Hospitalization afforded her relief from depressing circumstances of her worries. The sterility issue was investigated by Rubin's test and the tubes found to be patent. The patient left hospital abruptly following an emotional upset which she refused to discuss. During her stay of thirty-seven days she successfully lost 19 pounds in weight. The low blood sugar reading of 54 mgm. per cent at the end of a glucose tolerance test is of considerable interest in view of the enormous appetite, and deserves mention as a possible specific factor in the development of obesity.

In the last case, which is to be reported in another article on hypoglycemia, this factor of low blood sugar leading to gain in weight would again appear to be specific in certain types of tension states. In this case the tension is associated with a state of chronic anxiety and phobias.

Case 7—Patient was a 43-year old married housewife, admitted to the psychiatric clinic 11/27/35 in a condition of anxiety and mild reactive depression. This chronic anxiety state with phobias had made of her a semi-invalid since her marriage fifteen years before. Following the birth of each child she dreaded the increased responsibility, became depressed, developed numerous fears of crowds and high places, had outbursts of tension in which she clenched her hands, cried aloud. She spoke of definite episodes during which she felt smothered, dizzy, unable to sleep, and felt herself unable to find her way out of a store. In spite of all this she consistently gained weight, adding 47 pounds, because of her tendency to eat between meals to relieve her feelings of weakness. She described definite attacks of weakness and fainting which were always relieved by eating. Physical examination revealed only the obesity and a glucose tolerance test of 67, 95, 96, 61, 61, 61, and 64 at the end of five hours.

She is an individual with a poor heredity, her mother being a drug addict and her brother a nervous invalid. She dates her real illness to her honeymoon when she found herself sexually frigid. Her social life was a definite strain. Her first love affair ended in a serious disappointment and marriage was delayed a number of years. Sexual life was never satisfactory, the patient lying awake tense and restless following failure to have orgasm. She was timid, shy and sensitive but rarely showed it, had feelings of inadequacy, overconscientiousness, anxiety about her responsibilities. During 3½ months in hospital the anxiety and depression improved, but she constantly ate between meals and it was impossible to reduce her weight.

In a letter written three years later by her physician he stated that the attacks of hunger were greatly reduced in frequency and severity. The general psychiatric condition had changed very little and the weight had not diminished. As might have been expected, in view of the fact that the fundamental psychiatric disturbance had not been alleviated, she had gained ten pounds in weight.

Discussion

A series of cases of obesity have been presented in which the primary factors responsable seem to lie in the setting of the individual's personality make-up and reaction to life difficulties. In all of them the possible role of obscure physiological factors or fundamental somatic disease was excluded by thorough medical study. These cases, therefore, are likely to be of the more common variety of obesity problems confronting the average physician. They undoubtedly represent a special group of such obesity problems and they have been especially selected since the personality disturbance was immediately recognized by the referring internist. Nonetheless, they point to a need for a consideration of such factors in any obesity case. Particularly, if ordinary dietary regime proves unsuccessful, we feel that the physician should make a definite search for evidences of personality disturbance since this may give the clue to a more effective plan of treatment.

The first case was clearly that of a psychoneurotic individual wherein the obesity serves as a pivotal psychoneurotic factor. The second is a case of psychopathic personality with important life factors determining the overeating. The third is a case of depression with secondary weight gain; the fourth is a case of moodiness and depression giving rise to great desire for food. The fifth shows the role of protracted tension and insecurity in the increase of weight. The sixth case is that of a serious personality disturbance with a constant gain in weight. The final case is indicative of the specific role of hypoglycemia as a consequence of protracted anxiety and tension resulting in attacks of hunger, craving for food, and consequent gain in weight.

The multiple symptoms complained of clearly indicate the fundamental nature of the disturbance. Fatigue is prominent as a symptom and seems to be of the neurasthenic variety. While all but one of these cases was admitted to hospital primarily for the problem of weight reduction, some of them clearly recognized that the more pertinent disturbance was one of maladjustment and the obesity problem was incidental to the true nature of the personality difficulties themselves. In every case of obesity, therefore, it would be well to scrutinize the total personality make-up and type of reaction, since therapy may be doomed to failure unless the fundamental nature of the disturbance is understood.

Conclusion

The problem of obesity is one which sometimes involves the total personality in a rather sweeping manner. The successful treatment of obesity will in some cases depend upon the physician's thorough investigation of factors in the life other than the purely dietary or endocrinological.

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About Psychiatric Treatment

WARREN T. BROWN, M.D.
New Haven, Connecticut

Dr. Brown received his M.D. degree from the University of Texas in 1933. Following a residency in Houston, he came to New Haven Hospital in 1934, where he held the positions of interne, Assistant Resident Psychiatrist, Resident Psychiatrist, and Associate Psychiatrist. In 1935, he also became connected with the Yale School of Medicine, where he served in several capacities. Dr. Brown is now Physician-in-charge of the Psychiatric Clinic of the New Haven Hospital, and Assistant Professor of Psychiatry and Mental Hygiene at the Yale University School of Medicine. Dr. Brown is a member of the American Psychiatric Association.

Special schools and special methods of psychiatric treatment have been so prominently in the foreground for the past several years that the average practitioner may find the problem of treating even the simplest psychiatric condition veiled in a maze of theory and terminology. Hence it may be helpful to discuss a few simple fundamental principles which seem to underlie most of the superstructures of the more complicated psychotherapeutic systems.

Basically, psychiatric illnesses are those which express their major symptomatology through the behavior of the patient. That is, we arrive at the impression that we are dealing with an illness of a psychiatric nature by roughly the same technique that we decide whether our dinner guests are enjoying the steak. What is said, how it is said, what is done, how it is done, and behavior in general all lead to our conclusion. In much the same way we arrive at a diagnosis of a psychiatric illness. While there are many supplementary diagnostic methods now applied by psychiatrists, when we eliminate those conditions resulting from syphilis and those, "symptomatic psychoses," arising secondary to and as a result of other pathological disturbances, such as febrile diseases, trauma, brain tumors, etc., the rest of our diagnosing is based on behavior. This leads to a natural tendency to think of the symptomatology as the illness, and since that symptomatology consists of behavior, which is the medium of exchange of common logic, it seems to follow that psychiatric illnesses should respond to common logic. The fact that they do not supports the principle that symptoms themselves do not constitute disease, but result from it. This fact should never be lost sight of by the physician who is utilizing one of those forms of treatment which fall under the general classification of psychotherapy.

Many psychiatric illnesses result from the response of a specific person to a specific situation, but it does not follow that a complete alteration of the situation will remove the illness. A burn is not healed by putting out the fire that caused it, or removing the patient from it, although the latter is certainly a basic part of the treatment. It is also of importance to relieve a psychiatric patient from the effect of any environmental situation which has played an etiological role in the illness or has otherwise contributed to it, but this must be considered only an initial step in the treatment.

The treatment itself must primarily take into consideration the specific qualities of the patient who is to be treated. It is well to begin with the anatomy and first detect and eliminate or alleviate such pathology as may play a role in the illness or impair the treatment. Also, the general bodily capacities must be weighed as the degree of strength and drive demonstrated may determine whether you recommend mountain climbing or stamp collecting as a means of recreation and diversion. A second quality and one which should in every case be considered before treatment is undertaken is intelligence. Too frequently many hours of a doctor's time are wasted in a well-meant attempt to relieve a psychiatric illness by a system of explanation and reeducation which is so far above the patient's intellectual head that he either misses it all, or finds himself in possession of a chaotic jumble of meaningless though pleasant sounding words which immediately become incorporated into the symptomatology. Another factor which greatly aids in the therapeutic handling of a psychiatric illness is a thorough understanding of the patient's temperament and the role it plays in the patient's patterns of behavior. It is most probable that it will play a similar role in the treatment situation. Finally, an understanding of the patient's own opinion of himself and his environment and of these in relation

to each other is the best taking-off point from which to enter into the treatment.

Regardless of the many fascinating names that are applied to different forms of psychiatric therapy, the simple elements of human experience involved in all of them remain approximately the same. We may discuss these elements under five major heads. These are *Reassurance, Encouragement, Persuasion, Suggestion,* and *Re-education.*

Reassurance is probably most basic, and frequently plays an important role in treatment although it may not be recognized by either patient or doctor. Such reassurance may arise merely out of the patient's realization that the doctor looks upon him simply as a person with an illness and suggests that his is a natural and not unusual difficulty instead of something rare, frightful, and mysterious. Out of this reassuring feeling that the doctor understands, grows that priceless confidence in the physician without which any type of treatment suffers.

Encouragement becomes an important factor once the treatment is begun and the patient becomes disappointed that recovery is not immediate and is ambitendent about continuing the ordered schedule.

Although *persuasion* is frequently misdirected and futilely wasted, it is an indispensable therapeutic element. Time spent in attempts to persuade a schizophrenic that his delusions are not real may net the therapist nothing but fatigue; however, earnest persuasion may frequently result in convincing a patient to follow a certain valuable treatment regime or cease a damaging one.

Suggestion in therapy ranges all the way from the impressive interior decoration of the doctor's waiting room to the utilization of deep hypnosis. It is beyond doubt an active element in many of the elaborate psychotherapeutic systems and is responsible for much of their success. One need only turn to the noteworthy responses to placebos, shrines, charlatans, sterile hypodermics, and highly advertized mineral waters for evidence of the effectiveness of suggestion. It must also be kept in mind that the ethical authenticity of a particular treatment in no way deprives it of its suggestive value. Credit may frequently be granted elsewhere when results have inadvertently been due to suggestion.

Re-education, when used as a therapeutic approach, is not free from the influence of the foregoing elements. It consists chiefly in utilizing the patient's intelligence in establishing goals and purposes more in keeping with his needs and capacities than those which have led to difficulty.

The frequency with which psychopathology occurs in the average run of cases seen by the general practitioner testifies to the importance of his making himself familiar with the implications, development and prognosis of its various forms. Thus fortified with some knowledge of what he is treating, a sincere and logical application of the above-mentioned simple elements is likely to avail him more satisfaction and success than any attempt to apply hastily learned formulae from any complicated psychotherapeutic method.

NORTHERN CALIFORNIA SOCIETY OF NEUROLOGY AND PSYCHIATRY ORGANIZED

Neuropsychiatric Society of San Francisco which has not been active for the past several years, has recently been reorganized under the title of the Northern California Society of Neurology and Psychiatry. At a recent meeting of the Society, Dr. Walter F. Schaller was elected President; Dr. O. W. Jones, Jr., Vice President; Dr. Percy Poliak, Secretary-Treasurer. Dr. George Johnson, Dr. Paul Gliebe, Dr. J. Kasanin and Dr. Mary E. Mathes were elected as Councillors. The meetings will be held every two months in the various psychiatric institutions in San Francisco as well as through northern California. One of the aims of the Society is to bring into closer relationship the psychiatric work done in the various hospitals in San Francisco and in the Bay Area with the work done in the universities and in the State hospitals. The first meeting which was held in April was addressed by Dr. Walter Treadway of the United States Public Health, now attached to the University of California Medical School, who spoke on the National Program in Psychiatry and Dr. Aaron Rosanoff, Director of Institutions, who spoke on the State Program in Psychiatry.

A Study of the Vascular Tension in 599 Necropsied Cases of Vascular Encephalopathy

CULLEN WARD IRISH, M.D.
Los Angeles, California

Dr. Cullen Ward Irish was graduated from the Ohio State University, College of Medicine at Columbus, in 1915. He is an associate professor of neurology and psychiatry at the College of Medical Evangelists at Los Angeles. He is a fellow of the American College of Physicians and a member of the American Psychiatric Association. He has contributed many valuable papers to the medical literature.

In an intimate, clinico-pathologic survey of 1,000 cases of vascular encephalopathy found at 12,503 necropsies, done at the Los Angeles General Hospital, an investigation was made of the incidence of the contributory factors and associated conditions present. A study of the blood pressures reported in the protocols was included to demonstrate the degree of cardiovascular tension present in correlation with the various types and sites of cerebral vascular pathology.

Essential hypertension was considered to be the causative factor in 7.6 per cent of the 419 patients suffering fatal cerebral hemorrhage.

Blood-pressure recordings in this study demonstrated that thrombotic processes occurred in the presence of marked vascular hypertension and cerebral hemorrhage was found where the tension was subnormal, the average of readings in 262 patients suffering cerebral hemorrhage being 171/103 and practically equal to the average of 167/97 in 251 patients in whom cerebral thromboses had occurred. The average blood-pressure in 86 patients with embolic cerebral processes was 165/79, the highest recorded being 270/180 and the lowest 85/55. In 75 per cent of these recordings the systolic pressure was 85 to 150 mm. of mercury. On closer study of the comparative blood-pressures in the presence of thrombotic and hemorrhagic cerebral processes, however, higher tensions were found more frequently where cerebral hemorrhage had occurred. In 48.2 per cent of the 301 recordings in 262 cases of cerebral hemorrhage, the systolic blood-pressure was over 180 mm. of mercury. In 32.4 per cent of the 290 blood-pressure records of 251 patients in whom cerebral thrombotic lesions were found, the systolic pressure was over 180 mm. Hg. In the presence of cerebral hemorrhage 12.3 per cent of the systolic pressures were 240 to 300 and in but 4.1 per cent of the cases with cerebral thrombosis was this degree of hypertension present. The highest blood-pressure recorded in the hemorrhagic cases was 300/160, the lowest 70/50. Of the recordings in the patients with cerebral thromboses, the highest reading was 300/150 and the lowest 60/40.

A condition which is frequently overlooked or about which often no information is available is the *relative change* in the blood-pressure occurring immediately, or after some varying period of time, before the ictus. In the presence of hypertension noted following the cerebral vascular accident, it is sometimes assumed this was the usual degree of increased vascular pressure in the patient for some time. Ofttimes, however, where the patient had been attending the outpatient clinic or had recently been a hospital patient because of symptoms of cardiovascular renal disease or some unrelated condition, marked hypertension may have been recorded much in excess of the still abnormally high pressure found present on his admission following the apoplectic stroke. Or a decided decrease in pressure to within the limits of normal may have occurred.

Also, markedly elevated pressure may be found when it is known that the blood-pressure was normal but a number of weeks or few months previously, with no symptoms noted until the sudden catastrophe occurred. A history may be offered that hypertension has been present for some time and that evidence of a cerebral vascular accident was found during the night or early the next morning. The blood-pressure in these cases is liable to be found not above normal limits, or at least not excessively high. The drop in blood-pressure may have been but a relative decrease, leaving a still abnormal pressure, but this lessening in vascular tension appears

to serve as a definite contributory factor, and may be the only obvious immediate cause of the resulting thrombosis. On the other hand, in instances where it is known the pressure has not been elevated at a very recent date, a rapid increase may occur, cerebral hemorrhage present itself and the pressure again be found within normal range.

Hypertension must be recognized as a symptom, much as fever, resulting from many causes or a combination of several attributes. Hence, the far-reaching effects of a catastrophic accident, such as a sudden and devastating cerebral vascular insult, may be expected to decidedly alter the vascular pressure from its neatly balanced average or established necessary compensatory pressure caused by inelastic vessels, cardiac conditions, volume resistance, change in viscosity of the blood stream or whatever condition. Furthermore, cerebral centers which exercise coordinating control of cavity pressures, particularly those of intravascular pressure and intracranial pressure interrelationships and probably of the regulation of relaxation and contraction of the vessels, which is likely not entirely a local process, may leave their functions seriously impaired by the cerebral vascular lesion, directly or indirectly.

Consistent average differences in blood-pressure appeared in the presence of various types of cerebral vascular lesions, as might be expected, but also were demonstrated in regard to the different sites of the lesions. Where subarachnoid, subdural or petechial hemorrhage was present, the blood-pressures were uniformly not greatly increased and registered the lowest of the cases with cerebral hemorrhage, except where it had originated from the anterior cerebral arteries when the pressure was also comparatively low and twenty-four points below that found in the hemorrhages from other cerebral vessels. The average pressures were found to be increasingly high where the hemorrhage arose from the middle cerebrals, the cerebellar arteries and in the presence of multiple and bilateral hemorrhages, in the order named. Pressures were more markedly increased to over 200 systolic in the patients where the hemorrhage originated from the basilar artery and its branches, from the perforating branches of the middle cerebral arteries and from the posterior cerebral arteries. In the instances where multiple hemorrhagic processes were found it is presumptive that the causative factors had likely persisted for a longer period and when the hemorrhage resulted from perforating branches, usually large quantities of blood collected within the cerebral substance interfering with intracranial pressure relationships. The fact that the greatest pressures were present, however, when the hemorrhage came from the cerebellar, basilar and posterior cerebral arteries would seem to be of some significance. The intracranial pressure being increased in the posterior fossa by vessel ruptures in this region, where but little opportunity or space for expansion is available, in contradistinction to the inroads made upon the lateral ventricles by expansive conditions in the middle fossa, may be influential. Also, but a small hemorrhage in the limited available space in the posterior fossa would cause sudden pressure upon the medullary centers with involvement of many vital functions.

It is interesting to note that the average blood-pressures in hemorrhagic and thrombotic lesions in the supply of the middle cerebral perforating branches were the same. The *average* blood-pressure in the presence of cerebral hemorrhage from the various sources varied from 132 to 228 mm. of mercury while the plot curve of the *average* blood-pressures where thrombotic lesions had occurred was more flat, ranging but from 157 to 186. The highest pressures of those with thrombotic pressures were found in patients who had suffered multiple attacks, again indicating that contributory attributes in the production of both hypertension and cerebral thrombosis had existed longer than in the patients with single cerebral lesions and less hypertension.

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Tables published on pages 252-253.

93 Blood-Pressure Recordings in 86 Patients with Cerebral Emboli

Highest 270/180 *Average 165/79* *Lowest 85/55*

Systolic 85 - 270			*Diastolic 0 - 180*		
Systolic	*Number*	*Percentage*	*Diastolic*	*Number*	*Percentage*
85-120	32	34.4	0- 65	26	28.0
121-180	38	40.7	66- 85	28	30.1
151-180	16	17.4	86-105	28	30.1
181-200	3	3.2	106-125	7	7.5
200-270	4	4.3	126-145	1	1.1
			146-180	3	3.2
	93	100.0		93	100.0

290 Blood-Pressure Records in 251 Cases of 351 Patients with Cerebral Thrombosis

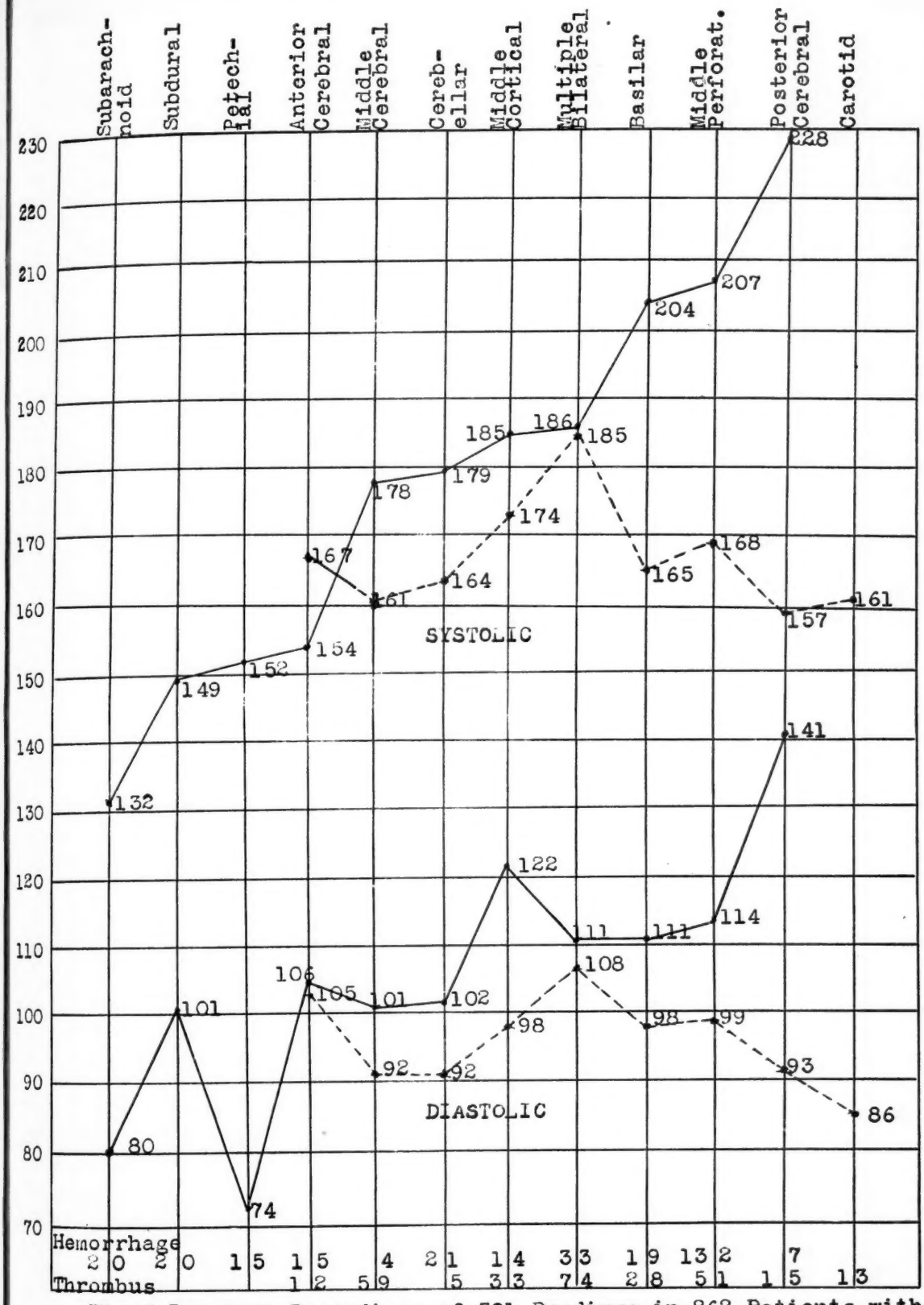
Highest 300/150 *Average 167/97* *Lowest 60/40*

Systolic 60 - 300			*Diastolic 0 - 200*		
Systolic	*Number*	*Percentage*	*Diastolic*	*Number*	*Percentage*
60-100	10	3.5	0- 65	23	7.9
101-120	25	8.6	66- 85	79	27.2
121-150	72	28.3	86-105	90	31.
151-180	79	27.2	106-125	57	19.7
181-200	35	12.1	126-145	22	7.6
201-240	47	16.2	146-180	17	5.9
241-300	12	4.1	181-200	2	.7
	290	100.0		290	100.0

301 Blood-Pressures in 262 Cases from 419 Patients with Cerebral Hemorrhage

Highest 300/160 *Average 171/103* *Lowest 70/50*

Systolic 60 - 300			*Diastolic 0 - 200*		
Systolic	*Number*	*Percentage*	*Diastolic*	*Number*	*Percentage*
60-100	8	2.7	0- 65	16	5.3
101-120	28	9.3	66- 85	61	20.3
121-150	50	16.7	86-105	81	26.9
151-180	67	22.2	106-125	69	22.8
181-200	41	13.6	126-145	36	12.
201-240	70	23.2	146-180	36	12.
241-300	37	12.3	181-200	2	.7
	301	100.0		301	100.0



Blood Pressure Recordings of 301 Readings in 262 Patients with Cerebral Hemorrhages and 290 Readings in 251 Patients with Cerebral Thrombosis: With the Average Pressures in the Various Vascular Involvements.

Thrombus ------ Hemorrhage_____

Newsnotes

FROM THE ANNUAL REPORT, VETERANS' ADMINISTRATION

In the annual report released by the Administrator of the Veterans' Bureau for the fiscal year ending June 30, 1939, relative to hospital and domiciliary care, the following information regarding psychiatric hospitals and beds was released. The total hospital load of the Veterans' Administration during the fiscal year of 1939 was 54,117 as compared with 50,899 on June 30, 1938; an increase of 3,218 or approximately 6.32 per cent during this period. Of that number, 1,930 were for neuropsychiatric cases. Of the total number of patients in the hospitals, 57.68 per cent were for neuropsychiatric diseases. Admissions for 1939 were the highest of any fiscal year to date. Since June 7, 1924, 1,206,966 or about seventy-five per cent of all admissions, have been for the treatment of disabilities not connected with service. Over ninety-two per cent of the admissions this year were on account of non-service—connected disabilities. During this year 9,372 were for psychotic or mental diseases and 13,240 for other neurological disorders. Of the deaths which occurred during the fiscal year, from a total of 10,880 deaths, 1,568 or 14.41 per cent were for neuropsychiatric diseases.

The present expansion program contemplates the acquisition within the next decade of 14,232 beds of which 5,268 will be for neuropsychiatric patients.

FAIRFIELD STATE HOSPITAL

Work at the Fairfield State Hospital, Newtown, Connecticut, is reported 95 per cent completed with nine new buildings scheduled to be ready for occupancy in July. These buildings are a part of the State's building program. The hospital, which has had 500 beds, will, with the new building, be increased to 2,000 beds.

KILLED IN ACCIDENT

Dr. William A. Deerhake, Superintendent of the Central State Hospital for criminal insane, Waupun, Wisconsin, was killed when an automobile he was driving, crashed into a truck load of W.P.A. workers. Dr. J. F. Klepfer, assistant superintendent of the hospital, has been appointed acting superintendent of the institution.

ADDRESSES MEETING

Dr. Raymond S. Crispell, Professor of Neurology and Psychiatry at Duke University School of Medicine, discussed "Neuropsychiatry in the Aged," before a meeting of the University of Virginia Medical Society on April 1st.

ADDRESSES STATE MEDICAL MEETING

Dr. Ernest M. Hammes, Professor of nervous and mental diseases, University of Minnesota and chief of the Neuropsychiatric service at Ancker Hospital, St. Paul, Minnesota, was a guest speaker before the 62nd annual session of the Medical Association of Montana.

Dr. Hammes delivered lectures on "Craniocerebral Injuries" and "Modern Treatment of Psychiatric Disorders."

ADDRESSES MEETING

Dr. Louis J. Karnosh, Associate Professor of Nervous Diseases at Western Reserve University, Cleveland; and Associate Editor of *Diseases of the Nervous System* was the guest speaker before the meeting of the Ohio County Medical Society held at Wheeling, West Virginia on April 19. The subject of Dr. Karnosh's paper was "Tumors of the Neck."

SOCIETY MEETS

Twenty-five members of the Minnesota Society of Neurology and Psychiatry, attended a scientific program at Rochester, Minnesota on May 11.

The morning program at St. Mary's Hospital was followed by a luncheon at the Mayo Foundation House, and the guest speaker was Dr. Peter Bassoe of Chicago, Associate Editor of *Diseases of the Nervous System*. His topic was "Some Neuropsychiatric Observations in Mexico, Puerto-Rico and the Virgin Islands."

Book Review

NEUROLOGY. By S. A. Kinnier Wilson, The Williams & Wilkins Company, Baltimore, 1940. 2 Volumes—1862 Pages—$21.00.

The long-awaited *Neurology* of the late S. A. Kinnier Wilson has finally been released to the medical world. Perhaps no other neurological work in recent times has evoked so much anticipation, nor is it likely that one will have lived up to its expectations as fully as this. Known to medical men the world over as the first describer of what later became known as Wilson's Disease (Progressive Lenticular Degeneration), the author, for many years prior to his untimely death in 1937, enjoyed the distinction of being one of the most brilliant and articulate clinical neurologists in England. The present work, respectfully edited and arranged by A. Ninian Bruce, is indeed a fitting memorial to a great neurologist.

Most neurological works have one of two faults: they are either so elementary as to be useless to any but the medical student, or they are so involved and obscure that they interest only the intensive student. Wilson's *Neurology* avoids both pitfalls and presents a comprehensive critical analysis of present day neurology; stimulating to the student, informative to the physician, and useful to the neurologist. The book is written in the effortless and lucid style of a scholar. Never obscure or pedantic, the text is studded with examples of Dr. Wilson's brilliant prose. From the wealth of his encyclopaedic knowledge he has supplied the reader with interesting and little known facts about neurologic disorders. His clinical descriptions are masterful and the patient almost comes to life before the reader's eyes. These descriptions are enhanced by many excellent illustrations of clinical and pathological specimens drawn largely from the material observed at the National Hospital, Queen Square. Perhaps no man had a greater acquaintance with the neurological writings of all lands than did S. A. K. Wilson and this storehouse is happily preserved for us in his long but carefully selected list of references. Treatment, always a weak spot in most works on neurology, is amply discussed under each disease heading. The American reader will be surprised to find that a therapeutic aid such as Vitamin B_1 in the non-deficiency neuritides is not even mentioned. Some of the newer developments such as electroencephalography, dilantin in epilepsy, and trigeminal tractotomy are also omitted. It is hoped that these minor omissions will be corrected in future editions.

In its emphasis on the purely clinical aspects of neurology this book fills a long felt need. No investigational work in medicine can have any validity unless it is based on thorough knowledge of the patient and his illness. This aspect is pounded home with all the considerable art of which the author was capable. Wilson's work will remain for many years the crowning triumph of the English school of clinical neurology.

Z. M. LEBENSOHN, M.D.
Washington, D. C.

Newsnotes

NEUROPSYCHIATRIC SOCIETY OF VIRGINIA MEETS

The June meeting of the Neuropsychiatric Society of Virginia was held at St. Albans Sanatorium, Radford, Virginia, June 19th, 1940.

The following program was presented:

1. "The First 100 New Admissions to the Petersburg State Colony" by *Dr. D. L. Harrell, Jr.*, Petersburg, Va.
2. "An Interpretation of the Drawings Made by Maladjusted Children" by *Dr. James N. Williams,* Richmond, Va.
3. "Experiences with Insulin and Metrazol in the Treatment of 80 Patients" by *Drs. John C. Palmer* and *James P. King,* Radford, Va.
4. "Some interesting Neurological Cases" by *Dr. Frank A. Strickler,* Radford, Va.

DR. ADOLF MEYER HONORED

Northern California Society of Neurology and Psychiatry honored Dr. Adolf Meyer at a dinner given in his honor in the St. Francis Hotel on Friday, June 14, 1940. Some seventy-five members and friends of the Society, distinguished Hopkins alumni, were present at the dinner.

HERE'S WHAT THEY SAY!!

A Few of the Unsolicited Comments from General Practitioners on Our First Issues

"This is the first journal on neurology which I could read and understand."

"I enjoyed reading the first issue very much and might say that if the magazine continues to present papers in such a readable and understanding manner as shown in the first issue, it will most certainly be a success. Most articles written on neurological subjects are too highbrow for the average practitioner like myself, but I notice that all the articles in this issue were very well written in plain language."

"It would seem that throughout the country the specialty of neurology is coming into its own as indicated by this new journal in the field."

"I have read the new journal from cover to cover and feel that it is filling a long-felt need in our profession. I have often wondered why psychiatric literature had to be presented so abstractly that only the intellectually elite could understand it."

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